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VOLUME II.

DISEASES OF THE ORGANS OF LOCOMOTION.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE ORGANS OF RESPIRATION.

CHRONIC CONSTITUTIONAL DISEASES.

INFECTIONS AND EPIZOOTIC DISEASES.

PATHOLOGY AND THERAPEUTICS

OF THE

DOMESTIC ANIMALS.

BY

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TRANSLATED FROM THE MOST RECENT EDITION WITH ANNOTATIONS

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TOGETHER WITH

*SELECTIONS FROM THE NOTES OF THE FRENCH TRANSLATORS,
AND ALSO FROM THOSE OF PROF. TRASBOT.*

VOL. II.

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PATHOLOGY AND THERAPEUTICS

OF THE

DOMESTIC ANIMALS.

SECTION I.

DISEASES OF THE ORGANS OF LOCOMOTION.

MUSCULAR RHEUMATISM.

THE word *rheumatism* has for a long time had a very comprehensive signification, serving to designate collectively all diseases of the organs of locomotion which are occasioned by *cold*. These diseases have in common the symptom of intense erratic pains, leading to more or less pronounced troubles of locomotion. They may affect the muscles, aponeuroses, tendons, tendinous sheaths, periosteum, and the articular synovial membranes, and produce lesions upon these organs the identity of which is not at all apparent. The grouping of these troubles has been perpetuated mainly on account of the general term which has been applied to them. In departing from this tradition, it would be possible to classify in this group a large number of other diseases à *frigore*, such as peritonitis, pleurisy, rheumatismal colic, etc. We shall not discuss the degree of relationship which exists between these morbid conditions; the diversity of causes, however, which are capable of producing them is a serious argument in favor of their differentiation. All, indeed, are not due to cold, and some of them appear to be of infectious origin.

On account of their etiological and clinical differences, it is proper to describe separately true rheumatism à *frigore*—*muscular rheumatism*—and pseudo-rheumatisms, especially *articular rheu-*

matism, which is an infectious disease. Muscular rheumatism is a myositis (*myositis rheumatica*), while articular rheumatism is an inflammation of the synovial membranes (*polyarthrititis serosa*). These affections have in common only the pains and troubles of locomotion by which they are accompanied. In our animals their manifestations are more obscure, their forms are more varied, and their diagnosis much more difficult than in man. Sometimes muscular rheumatism is not accentuated by any objective symptom.

The disease in young animals formerly designated *paresis* (Lähme) may be produced by articular or muscular rheumatism, and by several other morbid processes (see Pyemic Arthritis of Young Animals).

Etiology. Rheumatismal myositis is particularly frequent in the equine, bovine, and canine species; it is more rare in the ovine and porcine species. It is a common disease during the cold season. It is especially due to cold and dampness, cold winds and draughts, badly kept stables, damp pastures, sudden chilling of the overheated body, very cold baths, being out during cold nights (sheep), damp and cold kennels, hunting in swamps and the crossing of bodies of water by swimming (dog). Young and delicate animals are predisposed to it (lambs of the Merino race); the same is the case with those remaining too long in the stable (*stable rheumatism*), or such as receive too much food in proportion to the work they perform (*rheumatism of alimentation*); this last etiological circumstance acts especially in very fat dogs. A first attack predisposes to a return of the trouble.

The action of cold is still unknown; it is probable that the irritation which is produced by this agent upon the sensitive nervous ramifications, reflected on the vasomotor and muscular nerves, determines circulatory troubles as well as modifications of the chemical composition of the striated fibres—phenomena leading to confirmed myositis. This explanation is based upon the physiological fact that the organic mutations are considerably influenced, even while remaining in a normal condition, when the cold makes its action felt upon the skin.

At the present day it is no longer possible to consider the cessation of the cutaneous perspiration—a consequence of cold—as the cause; this has been said, by its arresting the elimination of the products of tissue waste, to result in a dyscrasia ultimately producing inflammation of the muscles. The hypothesis of an increase

of the *lactic acid* production is no more to be maintained than the preceding theory. Experimental suppression of the cutaneous functions (by varuishing), and the artificial introduction of lactic acid into the muscles, have never produced muscular rheumatism. The condition under consideration cannot be regarded as a pure neuropathic process, for in many cases the muscles present evident inflammatory alterations. On the other hand, it is certain that under the term muscular rheumatism have been confounded other neuralgic, traumatic, or circulatory troubles showing myositis. Excess of work and functional over-activity of muscles may determine alterations in these organs (traumatic myositis) which are similar to those of muscular rheumatism. These two processes are expressed by similar manifestations, and their differential diagnosis is at times difficult, but their nature is radically different.

Anatomical alterations. When the disease has been benign, the affected muscles do not show any alteration. Hyperemia and inflammation, which are hardly to be observed during life, are no longer to be found in the cadaver (the same fact is observed in slight catarrhs of the mucous membranes, the lesions of which disappear after death). In more serious cases we observe all the symptoms of myositis: hyperemia, hemorrhages, a serous exudate in the interstitial connective tissue, softening, discoloration, and segmentation of the muscular fibres.

In the horse, Bruckmüller has found the intermuscular connective tissue infiltrated with serum and made dense by a gelatinous exudate; the muscular fibres were pale and had lost their transverse striation. In a dog, Siedamgrotzky has seen all the aponeuroses infiltrated, softened and marked with red ramifications; the muscles were of a clear red tint and their capillaries were injected. In another dog affected by chronic rheumatism of the masticating muscles, he found an intense chronic interstitial myositis. When the rheumatismal process is prolonged, the inflamed intermuscular connective tissue becomes the seat of a cellular proliferation which leads to the formation of *rheumatismal callosities*; these are fibrinous connective tissue bands occupying the muscular interstices.

Symptoms. A. IN THE HORSE. In this animal the symptoms are ordinarily located in certain muscular groups, and preferably those of the extremities. It is quite rare to observe a generalized rheumatism. The patients suddenly take an abnormal extended position; the affected region is stiff; in cases where the

rheumatism is located upon the extremities they cannot rise without pain, and move backward with difficulty, taking very short steps, which do not compel them to flex the bony rays; the movements are sometimes accompanied by crepitant bruits which are produced in the articulations. The affected muscles are ordinarily sensitive, tumefied, and hard; sometimes the neighboring tissues are invaded by an cedematous tumefaction.

The most salient phenomenon of rheumatism is the migratory character of the trouble, which passes abruptly from one member to another. The marked tendency to a return of the disease, and a sudden improvement or complete disappearance of symptoms after more or less prolonged exercise, are also indications which are proper to rheumatism. It will be observed that the muscles of the shoulder—rheumatismal lameness of the shoulder (*omodyn timerica*, and of the lumbar region (rheumatismal sprain of the back, lumbago)—are mostly affected. In rheumatism of the shoulder the movements of the member are less extended in height and in development (pegged shoulder); in moving backward the foot is dragged upon the ground. The lameness becomes more marked when the animals ascend a hill or move upon soft ground; if the extensor muscles are particularly affected it increases when the animals turn short or wheel around. Lumbago is marked by a stiffness of position, sensitiveness and weakness of the lumbar region, a dragging walk, difficulty in rising, and sometimes by a *pseudo-paresis* of the hind quarters and falling. In cases where the muscles in the neighborhood of the coxo-femoral articulation are affected (rheumatismal lameness of the hip), the gait is slow and dragging; the posterior member moves with difficulty and covers little ground, the flexion of its rays is abrupt, and, as it were, spasmodic. It is impossible to know if there exists any intercostal muscular rheumatism in the horse (*pleurodyn timerica*) similar to that of man. If this localization exists it is not marked by any apparent symptom.

In cases of generalized rheumatism we observe, besides these manifestations, a more or less intense fever (we have noticed a temperature of 40.1° C.), also an acceleration of the pulse and breathing. Localized rheumatism is not accompanied by any febrile reaction; the pulse is but slightly accelerated and is sometimes hard. The serious forms sometimes coexist with other diseases à *frigore*, with respiratory or digestive catarrhs, colic, pleurisy, diseases of the ten-

dons, foundering, and more rarely with articular rheumatism. This complex pathological state is marked by a symptomatology much more alarming than that of rheumatism, and often leads to death.

The evolution is often acute, the duration short, and resolution very abrupt. The cure occurs at the end of a few days, within a week at the latest; as a rule, however, there is a tendency to a return of the trouble. In some cases muscular rheumatism passes into a chronic state; it then constitutes a very obstinate trouble, extremely difficult to combat, and which persists for weeks and months. A great many cases of lameness of the shoulder, of the hip, and paresis of the hind quarters, commonly designated *sprain of the back*, are determined by this form of affection.

B. IN THE OX. Muscular rheumatism is here marked almost in the same way as in the horse, but complications of articular rheumatism are more frequent. The regions mostly affected are the shoulder and loins; it may also be localized upon one or several members, or extended to all the muscles of the body. In the latter case the patients remain in an immobile position; the movements of the neck, shoulders, and extremities are difficult and laborious. The affected muscles are distended, stiff, and painful. The affection develops slowly. It often assumes a chronic type.

C. IN THE DOG. The rheumatic process is ordinarily localized in the muscles of the neck and shoulders, the back, and the psoas. It is rarely generalized. Its principal symptom is pain; the patients express it by complaints or actual cries; they fear the least movements and the lightest pressures, even a touch; some howl with pain as soon as we extend the hand to make an examination. Manœuvres performed with the object of making them rise—lifting the head, or extending the extremities—produce intense suffering; in some cases palpation of the psoas determines fearful pain (*lumbago rheumatica*). The expression is anxious, the neck is straight and extended, the movements are very difficult, the gait is stiff; the animals preserve an immobile posture or remain constantly lying down. It is especially hard and often entirely impossible for them to mount stairs. The sensitiveness of the various regions may become modified very rapidly; muscular groups which appear to be much affected in appearance recover at times their normal movements in a relatively short time, but others become affected, the rheumatism having only shifted its site. The appetite is generally

preserved, even in serious cases; if some animals eat little and slowly, it is due to the sensitiveness of the masticating muscles; and while we may often observe constipation, it is because the expulsion of excrements necessitates painful muscular contractions, and as no defecation takes place, constipation is produced.

D. IN THE SHEEP. The disease is sometimes localized, at other times generalized (stiffness, foundering). It is especially frequent in lambs. The gait is constrained, the legs have the "stiffness of stilts;" the neck, shoulders, and back are kept rigid, the head is often bent laterally; symptoms of lumbago are ordinarily observed. The affected muscles are hard and painful. The animals avoid all movement and remain constantly in a lying position. In general, the course is rapid, and a cure is produced within four to six days; in some cases, however, the affection is of long duration.

E. IN THE PIG. In this species muscular rheumatism is frequently associated with articular rheumatism. The extremities are stiff, the walk is painful and laborious (impeded), the dorsum is straight and rigid; gastric troubles often aggravate the disease, which is at times followed by a paretic weakness of the hind quarters.

Differential diagnosis. Muscular rheumatism may be confounded with various surgical diseases of the extremities: diseases of the foot, tendons, bones, and articulations (see treatises on surgery). It is clearly distinguished from articular rheumatism by the absence of the typical characters of the latter; the temperature is high, and the diseased articulations are tumefied and sensitive. When the affected joints are deeply located and hidden from sight and touch, the diagnosis is more difficult; in this case we must explore the different muscular groups and search for local rheumatismal symptoms (sensitiveness, hardness). The differential diagnosis between lumbago and spinal paralysis is sometimes embarrassing; the recognition of rheumatismal symptoms in another region, however, may direct us (see Diseases of the Spinal Cord). In the horse the manifestations of rheumatismal lumbago are similar to those of paraplegia produced by hemoglobinemia, which consists essentially of a rheumatismal myositis of the hind quarters. Hemoglobinemia and muscular rheumatism are but two degrees of one morbid process, the symptoms of which are more or less complex and severe according to the intensity of its causes and their mode of action. Muscular rheumatism may also coexist

or be confounded with pleurisy, nephritis, osteomyelitis, and with certain rheumatoid affections occurring during the course of some infectious diseases or accompanying bronchiectases.

Treatment. The principal means of treatment of localized muscular rheumatism are irritating and stimulating frictions made upon the affected region: camphorated alcohol, alone or mixed with essence of turpentine (1:10), oil of mustard (1:30), ammonia (1:15), etc. We must not forget that massage plays the principal part in the treatment of this disease, and that medical agents are only accessories. Priessnitz's compresses, cold douches followed by the application of very warm blankets, massage, and local sudorifics. In order to produce a profuse sudation we thoroughly wash the affected part with soap-water and cover the surface of the body with woollen blankets. Subcutaneous injections of veratrine are of great advantage for combating obstinate chronic rheumatism of the shoulder. In the horse we give hypodermatic injections of veratrine in this region (0.05 to 0.10 grammes) dissolved in alcohol (1 to 2 grammes); we increase the dose 1 centigramme each day; every fourth or fifth day we suspend the treatment for twenty-four hours. On account of the general excitement which is produced by this drug it is advisable to walk the animals as soon as the injection has been made. In the case of generalized muscular rheumatism we must resort to salicylic acid or salicylate of soda. In large animals we administer these agents in a dose of 25 to 50 grammes twice or three times a day; in the dog, pig, and sheep the dose is from 2 to 8 grammes per day. In general practice salicylate of soda is preferable to salicylic acid, which irritates the digestive mucous membrane and often causes inappetence. In large animals it may be given in electuaries or in pills; in small subjects it is better to administer it in an aqueous solution.

For the horse the following is the formula which we prescribe:

Salicylate of soda	100 grammes.
Licorice powder	25 "
Rye flour and water	q. s.

Fig.: The electuary is to be administered in three doses at three hours' interval.

For the dog and pig we dissolve 2 to 8 grammes of salicylate (according to the size of the sick animal) in 100 grammes of distilled water, and administer every hour a tablespoonful of the solution. Quite recently salol and naphthol have been recommended as substitutes for the salicylate of soda. Salol, which has

the same properties as salicylic acid, has the advantage over the latter of not irritating the stomach; we have obtained excellent results from it. The dose for the horse is from 15 to 25 grammes, for the dog 0.25 to 1 gramme; but the dog may be given as much as 4 grammes per day. Antipyrine, which is also recommended as an anti-rheumatic agent, is administered in the same doses as salol. We may also use evacuants, laxatives, or tartar emetic; this last is especially of advantage in cases of acute febrile articular rheumatism. In the dog we have used for a long time tincture of colchicum (5 to 15 drops twice or three times daily) as a light purgative and anti-rheumatic specific. We may also endeavor to produce an abundant sudation by external means (wrap the whole body with damp sheets, which ought to be covered with woollen blankets; the pig should be buried in manure up to the head), or by the administration of diaphoretics. Among the latter hydrochlorate of pilocarpine alone, in hypodermatic injections, produces sufficient effect upon the horse; it should be given in the following doses: adult horse, 0.4 to 0.8 gramme; colt, 0.2 to 0.3 gramme—dissolved in 5 to 10 grammes of water. Hübner has used with success pilocarpine (0.25 gramme dissolved in 4 grammes of water) in a nine-months'-old colt which was affected by muscular rheumatism; within two hours he observed perspiration, which continued for three hours. Upon three horses Siedamgrotzky did not obtain any successful result with subcutaneous injections of 0.2 to 0.4 gramme of pilocarpine, which was given for five days. It is, of course, understood that we must avoid any chilling of perspiring animals.

Finally, we must allow the animals rest; they should be placed in a stable free from draughts and be fed moderately.

ARTICULAR RHEUMATISM.

Articular rheumatism is a febrile infectious disease, which is accompanied by general troubles, and consists of an inflammation of one or several articulations (polyarthritis). Of all domestic animals the ox is most frequently affected by it; then come the horse, dog, and pig. Arthritic troubles of newborn animals constitute a complex morbid state, which ought to be separated from acute articular rheumatism. We shall give a description of it in the following article.

Etiology. Up to the present time the taking of cold has been considered as the principal cause of this form of rheumatism (draughts, damp cold, defective stables, etc.). But it is far more probable that, in our animals as in man, articular rheumatism is an infectious disease, and that cold intervenes in its genesis only as an occasional cause.

The following are the principal facts which establish its specific nature: 1. General febrile symptoms and the initial characteristic period of infectious diseases. 2. The simultaneous affection of articulations which are more or less distant from one another. 3. Endocarditis, which sometimes complicates the rheumatism. 4. Its appearance in model stables, where the taking of cold could not be considered as a cause. 5. The close relations (noticed by different authors, particularly by Auer) which exist between non-delivery of the cow and articular rheumatism. Auer found it especially in young cows (after the second or third calving); in two-thirds of the 67 cases observed by him the history enabled him to attribute the trouble to abortion or non-delivery; he considers the absorption of septic products of the uterus as the sole cause of articular rheumatism. Dinter has seen it existing exclusively in cows that had recently calved.

From these observations it seems apparent that the puerperal uterus of the cow constitutes the principal source of the specific infectious matter of rheumatism. Cold (draughts, damp stables, etc.) acts by favoring the penetration of pathogenic elements into the tissues and their pullulation in the organism.

The infectious agents of articular rheumatism are still unknown; but it seems that this disease may be determined by various micro-organisms. It constitutes a complex morbid process, especially from an etiological point of view.

Pathological anatomy. The principal alteration is serous synovitis; purulent synovitis is only found in exceptional cases. Generally, several articulations are affected. In the acute form (Harms, Leblanc) the synovial membrane is red, hemorrhagic, or thickened, tumefied, and strewn with turgescient villi, which are of a dark red color. The synovia is bloody or has a suspicious look; its quantity is considerably increased. The cartilages are of a rose tint at the beginning, later they become yellowish; their surface is velvety or rugous. The tissues which surround the joint are injected and overrun with interstitial hemorrhages and infiltrated

with serum (peri-arthritis); the connective tissue especially is the seat of a gelatinous infiltration; the adjacent muscles are softened and œdematous. The epiphyses and the marrow of the bones are red, congested, and ecchymosed. In the chronic form the synovial membrane becomes considerably thickened and covered with a very vascular connective vegetation (*arthritis pannosa*); the articular cartilages, which are affected by fatty degeneration, show ulcerous losses of substance and are eliminated by fragments. In some cases the arthritis becomes deforming, as in man. In a dog suffering from chronic articular rheumatism, Moore found the phalanges of an anterior member ankylosed between themselves and also with the metacarpus, and also covered with very irregular osteophytes.

The principal contingent alterations are due to complications of endocarditis, pleurisy, and peritonitis. All others are without any importance.

Symptoms. Articular tumefactions constitute the dominating symptom. In the majority of cases they appear abruptly, and in a little while—sometimes in one night—they acquire great size. They are generally observed in several articulations of one or more members, mostly upon the knee, hock, or stifle-joint; they are hot, painful, and fluctuating in certain places; the skin, which is distended over their surface, is red upon the regions which are bare of pigment; the inflammatory swelling is extended further than the joints; the neighboring organs (tendons, synovial sheaths) participate in the inflammatory process.

These articular lesions determine such very great lameness that the owners often erroneously attribute it to the existence of a fracture. The diseased articulations are the seat of a very intense sensitiveness; in exploring these or in making even very limited movements upon the bony rays by which they are formed, we produce fearful suffering; support by the affected members is lost. If in a standing position, they avoid the least displacement. Most animals stretch themselves upon the ground, complaining or groaning; they can only be made to rise with much difficulty. The general temperature is ordinarily from one to two degrees above the normal; the pulse is slight and hard; in the ox it beats from 70 to 90 times per minute (Harms).

As a rule, the appetite has disappeared, rumination is suspended, excrements are rare, the muzzle is dry; the lacteal secretion is diminished or exhausted, and the milk becomes acidulated and clots

very rapidly. In the ox, emaciation is often considerable as early as the third day. To these symptoms others are sometimes added, due to special complications. Acute endocarditis is not rare in the ox (Leblanc, Hering, Ruste, Mayer; see Endocarditis); we may also observe pleurisy, peritonitis (Harms, Leblanc), pericarditis (Leblanc), laryngitis (Derr), tendinous synovitis (Harms). These secondary troubles can only be explained by the infectious nature of the disease.¹

Articular rheumatism ordinarily assumes a chronic type; the prolonged acute form is exceptional. Sometimes, with the disappearance of the fever, the articular tumefactions become reabsorbed, and a cure may occur within two or three weeks, but this happy termination is extremely rare. The resolution is very often only apparent, and the return of the disease soon becomes manifest; the process leaves a very seriously affected articulation in order to attack another a few days later (rheumatic migrations). In the majority of cases the trouble becomes gradually aggravated. The febrile symptoms disappear, but the articular lesions persist, and with them the troubles of locomotion; the emaciation becomes every day more evident; in the cow, the milk secretion does not return; digestive troubles and gastric catarrh follow and a very intense and obstinate diarrhea appears; the muscles of the diseased regions become atrophied (muscles of the shoulder, etc.), the extremities are stiff, the joints are the seat of degenerating processes—they become *knotted*. This condition may last for several months. In a horse Dammann has seen rheumatism persist for eight months without improvement. When the animals are not killed they die from exhaustion; the body is covered with eschars produced by prolonged decubitus.

Prognosis. This is always serious in the ox. According to our observations it is much more favorable in the dog.

Treatment. The nature of the infectious agents which produce

¹ Mégnin has reported a curious example of ambulatory lameness which was observed in a thirteen-year-old horse, which had never before shown any signs of rheumatism. Great claudication was observed successively, on the same day, upon the left and right hind legs and also upon the left foreleg; "within a quarter of an hour we could observe this strange lameness affect successively three legs." In order to recognize its erratic character, it was sufficient to leave the animal at rest for a few minutes; it also became obliterated during exercise. A tumefaction of the fetlock was observed, which was especially marked upon the line of the great sesamoid sheath. Cardiac symptoms existed, besides a very strong precordial bruit, a quivering or a rolling bruit. (See Recueil Vét., 1888.)—N. D. T.

rheumatism not being known, the only prophylactic measure which can be given is to keep the animals from the action of cold. In the cow we must remove the placenta at the proper time and disinfect the uterine cavity carefully. The disease is generally combated with salicylic acid and salicylate of soda (daily dose of salicylate of soda for horse and ox, 100 to 150 grammes; for the dog and pig, 2 to 8 grammes). We may replace salicylic acid by salol, naphthol, and antipyrine (see Muscular Rheumatism). In order to induce reabsorption of the arthritic exudate and the elimination of infectious principles by way of the intestinal tract, we administer laxatives in large doses. Formerly tartar emetic was frequently used. As external treatment, we advise rubbing the diseased regions with carbolated or camphorated ointment and wrapping them carefully.

In the chronic form, when the exudate is stationary, we may try vesicant frictions (tincture of iodine, cantharides ointment, and ointment of biniodide of mercury). The patients should be allowed to rest and must be given a moderate alimentation. The stables ought to be well ventilated and kept warm.

PYEMIC ARTHRITIS OF YOUNG ANIMALS.

Paresis of Newborn Animals.

REMARKS ON THE TERM "PARESIS" (*Lähme*). Under the name *paresis* a series of diseases affecting newborn animals (colts, calves, lambs, young pigs, dogs) was formerly expressed, although presenting essential differences as to their nature and their causes, and having as a common manifestation locomotor troubles only. The term "paresis" is therefore a collective appellation which no longer deserves to be preserved with the signification which has been attributed to it. The necessity of replacing it by scientific expressions suitable to the different diseases which it has served to designate has been long felt by veterinary authors. As early as 1839, Träger states that *paresis of young horses* "represents quite a pathological vocabulary." Modern researches, especially those of Böllinger, have extended and confirmed our knowledge about this complex morbid state; they have permitted us to recognize in it:

1. *Pyemic arthritis*, which is the most frequent form (Böllinger); it is the consequence of a septic infection starting from the umbilical region.

2. *Fatty degeneration of the red muscles*, observed especially in young pigs.

3. *Acute articular rheumatism*.

4. *Acute or chronic muscular rheumatism*.

5. *Rhachitis* of young pigs.

6. *Tetanus* and *cerebo-spinal meningitis* in lambs.

7. *Consumption* (*Darrsucht*), a disease of young horses which is probably only an intestinal or ganglionic (mesenteric) tuberculosis (?).

8. Various other affections of sucklings, such as intestinal catarrhs, broncho-pneumonia, pleuro-pneumonia, general weakness, weakness of the extensors of the extremities with contraction of the flexors (colt, dog). Some of these diseases (muscular and articular rheumatism) have been described previously; others will be given in their proper places. Pyëmic arthritis could be studied at the same time as pyemia, but as it represents the larger number of cases of *paresis*, and as, on the other hand, this expression is too much in use to be easily suppressed, we have devoted a special article to it. We will also describe *fatty degeneration of the muscles* at the end of this article.

Etiology. Pyëmic arthritis is observed in the colt, the calf, and the lamb (Roloff). It is due to the introduction into the blood of putrid matter coming from the suppuration of the thrombus of the umbilical vein. This thrombosis is the consequence of suppurative inflammation developed upon the surface of the umbilicus and the putrefaction taking place in its neighborhood.

Infection of the umbilical wound by the products of putrid decomposition which cover the ground of the stables is the starting-point of these morbid processes. Quite recently Uffreduzzi has cultivated two microbes—a bacillus and a micrococcus—obtained from calves which had died of pyëmic arthritis. Inoculation of cultures has produced mixed symptoms belonging to pyemia and septicemia. These two infections, then, may occur as complications of the umbilical wound. An unclean condition of the umbilicus is the principal cause of the trouble. Böllinger mentions also as predisposing conditions: a thick and gelatinous umbilical cord, tumefaction of the umbilical vessels at the time of its laceration, its rupture at a point too close to the abdomen, contusions of the umbilical wound, traumatism of the abdominal walls, and, lastly, birth during the cold season. (A prolonged

sojourn of the animals in the stable increases the proportion of putrid matter in them, and thus favors the infection of the umbilical wound. The disease sometimes runs an enzootic course in flocks and stud farms, where it occasions considerable losses. In the national stud farm of Württemberg, during a period of fifteen years, out of 187 colts that died when sucklings, 85 succumbed to "paresis" (Hering).

Pathological anatomy. The alterations are those of suppurative polyarthritis and of pyemia; those of septicemia also are sometimes found. The cord and the tissues in its neighborhood are sometimes tumefied; at other times the umbilical wound is very much inflamed and covered with pus, its edges being occasionally the seat of an ulcerative process, and by compressing it purulent matter is made to escape from it. Besides this omphalitis (the exterior symptoms of which have at times disappeared), we find a deep umbilical suppurative thrombo-phlebitis, and even a thrombo-arteritis, which is accompanied by thrombosis of the portal vein and its hepatic ramifications. The articular synovial membranes are injected, thickened, and tumefied; the synovia, which is very abundant, is suspicious when mixed with flaky clots; later it becomes entirely purulent. The articular cartilages are ulcerated; the epiphyses may be necrosed. Peri-articular abscesses often produce a purulent wasting of the tendons and neighboring muscles; these are affected by fatty degeneration. We find metastatic centres in most of the organs and tissues (liver, lungs, brain, kidneys, muscles, and subcutaneous connective tissue); in the liver they are of the size of a millet-seed to a cherry or larger; in the lung they reach the size of a hen's egg; their color is dark red or yellowish. All tissues and all organs show at times inflammatory alterations; we may find lesions of the most varied forms, such as pleurisy, endocarditis, pericarditis, broncho-pneumonia, inflammation of the tendinous sheaths, peritonitis, purulent iritis, meningitis, etc. Lastly, the heart, liver, kidneys, and muscles have undergone fatty degeneration.

Symptoms. In the majority of cases pyemic arthritis occurs a short time after birth. Out of 67 sick colts observed by Hering on a stud farm, 47 (70 per cent.) died during the first three weeks of life. Böllinger found nearly this number on another farm (75 per cent.). No joint is exempt from rheumatism, but those which are mostly affected are the knee, hock, stifle, elbow, hip, and shoul-

der ; it is also frequently found upon the fetlock and coronet, also in the costal and intervertebral articulations. As a rule, the appearance of arthritic symptoms is preceded by certain general phenomena (fever, diminution of appetite). Articular tumefaction increases rapidly ; it is very painful, hot, and distended ; at the beginning it may become attenuated and disappear, later it has a tendency to abscess formation ; when this results the pus will more or less rapidly clear a way for its exit. Several articulations are almost always affected at the same time. The patients are very lame in one or several members ; they ordinarily take a decubital posture. Within a certain time diarrhea is added to these symptoms ; the fecal matter, which is of a grayish color, spreads a fetid odor ; diarrhea often alternates with constipation. Finally, cachexia and exhaustion appear. According to the localization of the metastases, we may observe the most varied and unexpected complications : pneumonia, sudden tumefactions in various regions, cerebral symptoms, blindness, etc.

The disease generally takes an acute type. Its average duration is two to three weeks, but there are cases where death occurs within a few days.

Prognosis. This is very serious. According to Hartmann, more than half of the affected animals perish ; Böllinger gives a mortality of 72 per cent. ; according to Darreau, in some countries it is said to rise to 90 per cent. Convalescence is very slow, and the animals rarely recover their former health. Articular tumefactions and stiffness of the extremities always take a long time to disappear.

Differential diagnosis. Pyemic polyarthritis must be differentiated from acute articular rheumatism. The following facts enable us to establish a diagnosis : The appearance of an articular tumefaction during the first days of life indicates pyemic arthritis, especially when at the same time we find a suppurative omphalophlebitis ; in rheumatism, abscess formation is so exceptional that we may, in all cases, consider it as the expression of a pyemic process. Articular rheumatism is, too, very rare in colts, which are generally kept from the influences which give rise to it (cold).

Treatment. From the pathology of pyemic arthritis, it follows that the treatment ought especially to be prophylactic. Böllinger advises to ligate the cord, to favor cicatrization of the umbilical wound by the use of antiseptics, and to apply upon the abdomen a

protecting bandage. If necessary, this latter may be replaced by a coat of tar. The stable must be kept perfectly clean, excrements and urine being removed frequently, and an abundant litter should be given. The disease must be combated by antiseptic applications upon the umbilical region (carbolated or cresol water; carbolated, cresol or iodoform ointment, 5 per cent.), and by the administration of antipyretics and antizymotics (camphor, subcutaneous injections of camphorated alcohol in a dose of 1 to 5 grammes). We may also use stimulants (alcohol, wine).

The treatment of diseased articulations is purely surgical; it consists in giving an outlet to the pus by means of a puncture or incision.

The expression of *fatty degeneration of the muscles* has been applied, in sucklings, to a disease of the apparatus of locomotion which is quite common in animals of the English breeds of pigs and also in lambs of improved breeds, more rare in colts and calves. Described by Fürstenberg,¹ Roloff,² and Repiquet,³ this muscular alteration, which often appears already in the second half of foetal life, is but an epiphenomenon of a general fatty degeneration; it must be studied in the article on Obesity.

Its causes are as yet little known. An hereditary predisposition has especially been charged—the mother's obesity (animals of precocious breeds). Its development is favored by a too abundant alimentation and permanent stabling of females in a condition of gestation. Fürstenberg mentions also consanguinity.

Anatomically it is characterized by anemia, fatty degeneration of the muscles, which have lost their morphological properties and present a lardaceous aspect, appearing as if they were cooked; by degeneration of the liver, kidneys, brain, pancreas, intestinal glands, etc.

Its principal symptoms are general weakness, slowness and hindrance of movements, inability to walk or to remain standing. In serious cases the patients are absolutely apathetic. We have seen death produced in coma, without having been announced by any alarming symptom; but at times it is preceded by diarrhea, spasms, and paralysis. In a certain number of cases it is caused by œdema of the lung. We can only intervene in a useful way by

¹ Fürstenberg: Virchow's Archiv, 1864.

² Roloff: Annalen Landwirtschaft, 1864.

³ Repiquet: Journ. de Lyon, 1888.

prophylaxis: in regenerating the blood by repeated crossings, and in giving exercise and a moderate amount of alimentation to females which are in a gravid condition. The treatment of affected subjects always remains without results.

OSTEOMALACIA AND RHACHITIS.

GENERAL CONSIDERATIONS UPON DISEASES OF THE BONES.

Bone diseases which belong to the domain of internal pathology—especially osteomalacia and rhachitis, which are the most important—might be described in the section on general nutritive troubles; however, as their principal symptom consists in a lack of harmony of the movements of the legs, it has been the traditional custom in veterinary medicine to classify them in the group of troubles affecting the apparatus of locomotion.

Pathological anatomy has thrown but little light as yet upon the pathogenesis of these affections, their causes are in fact too incompletely understood to enable us to establish a classification based upon the etiology. It has seemed to us rational to divide them into two large groups:

1. Diseases of the bones occurring in *young animals*, among which *rhachitis* is the most important.

2. Diseases of the bones appearing in *adult animals*, the principal of which is *osteomalacia*.

Whilst opinions are yet dissentient upon the relations existing between rhachitis and osteomalacia, a close relationship is demonstrated. We have observed for a long time that at certain periods they appear simultaneously in the same herd or upon animals in the same stable; it has also been recognized that their common pathogenic condition which has most importance is the want of calcareous salts in the food. On the other hand, experimentation has permitted us to produce simultaneously rhachitis and osteomalacial alterations upon the same individuals. Undoubtedly these alterations show very striking differences, but we must not forget that in a normal state the bony tissue is far from having a uniform composition or texture in young animals as well as in adults; age, therefore, may well be an important factor in this diversity of lesions. In considering rhachitis as a form of osteomalacia, due to special conditions of growth of the young bone, we may thus define these two diseases:

I. *Osteomalacia* is a softening of the adult bone, due to a re-absorption of calcareous salts.

II. *Rhachitis* is a want of consolidation of young bone, due to insufficient calcification caused by the want of calcareous salts.

EXPERIMENTAL RESEARCHES UPON THE ETIOLOGY OF RACHITIS AND OSTEOMALACIA. The numerous works which have been undertaken with the object of clearing the pathogenesis of these morbid conditions have given only incomplete results, but they have started three theories which may be thus comprised: the theory of *inanition*, the theory of *acids*, and the theory of *inflammation*.

1. In the *theory of inanition* it is admitted that an insufficient proportion of calcareous salts in food is the principal factor in both diseases. Among experiments contributing to give it some value, the most conclusive are those of Roloff and Voigt. In an adult goat and a sheep which were subjected to a regimen which was poor in salts of lime, Roloff has seen symptoms of osteomalacia produced; in experimenting upon young dogs and pigs, he has produced rhachitis characterized by lameness, incurvation and contraction of bones, tumefaction of the costal cartilages (rhachitic rosary), contraction of the pelvis, thickening of the articulations, formation of *double articulations* (*Doppelgelenke*), *bear paws* and *badger paws* (bending legs) the ox knee, crooked hock-joint, the bent knee, deformities of the spinal column, intra-periosteal fractures; laborious and staggering gait, general weakness, abnormal sensitiveness to touch, and symptoms of licking-disease. The same clinical appearances have been observed by E. Voigt in puppies subjected to a diet poor in calcareous salts. In these animals ossification was considerably delayed; besides a decrease in their thickness and diminution of their strength, the bones showed all the symptoms of rhachitis; also epiphyseal tumefactions, inflexion of the ribs and the scapula, incurvation of the extremities, a laborious, painful walk, or a complete suppression of movements.

To these first results, which were confirmed by Chossat, Milne-Edwards, and Lehmann, some authors have offered in opposition the negative facts of Weiske, who has seen his own experimental subjects succumb without having shown any rhachitic symptom. It has been observed with good reason that these animals perished, not through the fact of want of calcareous phosphates in food, but rather to inanition, and before any bony lesions had had time to

become developed. These negative results do not weaken the preceding in any way, which establish a possibility of experimental production of rhachitis. In addition, those obtained by Roloff and Voigt are confirmed by clinical observation. For a long time the want of salts of lime has been considered as a principal determining cause of rhachitis and osteomalacia; in numerous cases the insufficiency of these salts has been observed practically; very frequently, in impregnated and good milch cows, osteomalacia is the result of considerable withdrawals of calcareous matter which are necessitated either by the development of the fœtus or by an abundant milk secretion. Let us add that the administration of calcareous phosphates constitutes the most efficient treatment of these two diseases. Their development under the influence of a deficiency of lime salts in the organism, as demonstrated by experimentation, is, as can be seen, confirmed by practical facts.

2. *The theory of acids* is based on the solubility of calcareous salts in lactic acid. In cases of catarrh of the stomach or intestine—diseases which are particularly frequent in young animals—a very large proportion of lactic acid would be formed at the expense of the milk and carbohydrates ingested by the animals; this agent would pass into the blood and dissolve the calcareous salts of the bones, and, by irritating the bony tissue, would produce an abnormal cellular proliferation. Heitzmann thought he had demonstrated experimentally the correctness of this theory; by repeated hypodermatic injections of lactic acid and by an administration of this acid internally, he considered he had produced rhachitis in the dog. Roloff, Heiss, Arloing, and Trippier, who have repeated Heitzmann's experiments, have never obtained anything but negative results. Roloff thinks that in these experiments it is the want of calcareous matter in food and not lactic acid which is the cause of rhachitis (Heitzmann gave his animals food which contained no salts of lime). The assertions of some authors on the existence of lactic acid in the urine and in the bones of rhachitic animals are weakened by numerous facts. Virchow has constantly found an alkaline reaction of the new rhachitic bone; on the other hand, very exact analyses of urine have not shown the presence of lactic acid. Besides, in admitting that this last did pass into the vessels, it would undoubtedly become decomposed very rapidly into carbonic acid and water, where it would become combined with the blood bases. Let us also remark that the ingestion in a large quan-

tity of other acids (those of swill, for instance) has never produced rhachitis or osteomalacia.

Recent researches of Siedamgrotzky and Hofmeister have shown that, under the influence of a prolonged administration of lactic acid, the mineral substances diminish in the bones—without, however, causing the appearance of the characteristic phenomena of rhachitis or osteomalacia. From this they conclude that these last affections could not be produced by that acid; but this latter, however, possesses dissolving properties which have to be taken into account. According to these authors, lactic acid is said to play a certain part in the etiology of affections of the bones.

3. *The theory of inflammation* considers the two morbid conditions which are here concerned as inflammatory affections of the bony tissue, which are produced by an irritating substance circulating in the blood. It is based upon experimental verifications made by Wegner, who has seen, in some animals, a thickening of the diaphyses through prolonged administration of small quantities of phosphorus. Kassowitz concluded from this that there exists in the blood of rhachitic subjects a principle the effects of which are similar to those of phosphorus and which starts an inflammation in the bones; these organs would become the seat of an abnormal vascularization, the dilatation of their vessels would prevent the calcareous salts being deposited there, the dissolving action of the carbonic acid of the blood would make itself felt upon the mineral elements of the bones and produce the wasting of these organs. In repeating his experiments Wegner has seen the development of bony alterations which were entirely similar to those of rhachitis. They ignore entirely the nature of the irritating principle the action of which produces rhachitis or osteomalacia. The bone-alterations in syphilis of man permit us, however, to suppose, by analogy, that it is of an infectious nature (hematogenic osteitis). Virchow long ago asserted that osteomalacia of man is but a variety of parenchymatous osteitis. Veterinary authors have also suspected the inflammatory origin of these bone affections; formerly they were attributed to the action of cold. The sufferings by which they are accompanied caused them to be confounded with rheumatism, which, besides, complicates osteomalacia quite frequently. It is therefore not astonishing that cold has been charged as the cause, but in the immense majority of cases it does not at all intervene, and its mode of action is much less understood than that of the

infectious elements. If the theory of inflammation exists in the domain of the possible, nothing precise is known on the subject. In substance, osteomalacia and rheumatism represent special troubles of the nutrition of bone tissue. They may be accompanied by various complications, especially by rheumatism. The inflammation may have a certain influence upon their development, but the action of lactic acid is of no importance. New researches are necessary in order to enlighten us as to their etiology and their pathology.¹

In the actual state of science, the affections here considered must be considered as two pathological conditions which are related to an insufficiency of lime salts in the bone tissue—*calcareous inanition*. This fact has served us as a directing idea in compiling the following articles.

A. Osteomalacia.

FRAGILITY OF THE BONES: SOFTENING OF THE MARROW : OSTEOPSATHYROSIS.

Etiology. Osteomalacia is generally described under the denomination of "softening of the bones." It is quite frequent in the ox, but rare in the sheep, goat, and pig. In zoological gardens it has been observed in the giraffe and in certain birds. It was noticed in the ox by the oldest authors. Vegetius gives a brief description of it.

¹ Among the pathogenic circumstances which determine rhachitis, two are especially important: 1. Insufficient absorption of lime and phosphates. 2. The existence in the blood of an excess of acid, especially lactic acid. Lime and phosphoric acid do not appear to enter into the economy as lime phosphate; lime penetrates especially as a chloride, carbonate, or in combination with organic acid; phosphoric acid penetrates as an alkaline phosphate, very probably as phospho-glyceric acid. The latter, which is produced in the duodenum and absorbed in the intestine, can only be formed when there is an almost complete integrity of all the digestive functions; its production necessitates quite a high degree of gastric juice, a great alkalinity of intestinal juices, integrity of biliary and pancreatic secretions. Phospho-glyceric acid, which is formed in the intestine, would become combined with the lime within the organism, in order to form ossifying phosphate of lime. Lactic acid is produced in considerable quantities in the stomach and intestine under the influence of digestive troubles. It must exist in the economy of a large number of rhachitic animals. In opposing the precipitation of phosphate of lime in a tribasic state it may interfere as far as concerned in the want of calcification.

This last influence appears to be the principal pathogenic condition of osteomalacia. The osteomalacial bone has a constant acid reaction (lactic acid); thus the accumulation of acids in the bone tissue, in order to produce this reaction, creates a condition which is sufficient for dissolving and eliminating the ossifying phosphate of lime. (See Ch. Bouchard: *Diseases Produced by Diminution of the Nutrition*, Paris, 1882.)

There are countries in which it is stationary, and where, in certain years, it exists in an enzootic state; by its epizootic character it has, at different times, attracted the attention of their governments. It is also observed in a sporadic state on farms and agricultural centres. Milch cows seem to be predisposed to it; working oxen, old and young animals, are more rarely affected. Gestation and lactation favor its development and influence its evolution. The more abundant the lacteal secretion, the more rapid is its course; on the contrary, an amelioration is generally produced when lactation diminishes. In certain countries the cows are almost fatally affected six or eight weeks after calving. These circumstances have induced us to consider an abundant elimination of lime salts as a causal condition of the disease.

The want of calcareous salts in the soil and an insufficient quantity of lime in the food are both causes of osteomalacia. Swampy, turfy, and sandy soils of plutonic formation, which are poor in phosphoric acid (where nothing but carbonate of lime is found), favor or even cause its development; plants of damp pastures, especially gramineous plants of a poor quality which grow in such localities during very dry seasons—at which time the salts of lime are not dissolved by moisture and incorporated by the plants; food which is poor in calcareous salts (potatoes, turnips) act in the same manner. These facts enable us to understand why osteomalacia exists in preference in the stables of the poor, where the animals are fed on very voluminous food having little nutritive value, such as straw and all kinds of refuse. The absence of calcareous salts in the water of which the animals partake may also originate it.

Formerly it was considered that defective stables (especially damp, low stables having a moist odor) were causal; osteomalacia, however, could not be produced merely by these causes; they could at most favor its development when the animals are in an unfavorable state of nutrition. Let us add that weak subjects are predisposed to bone diseases. At the present time nothing positive is known concerning the influence of infectious principles.

The large majority of cases designated in our literature under the name of “fragility of bones” are but cases of osteomalacia, which are complicated or not by rheumatism. *Fragility of the bones* is, moreover, but a symptom which is also observed in other diseases, mainly in rhachitis. It must be related to these affections.

Pathological anatomy. The principal alterations are a de-

calcification, a softening and hyperemia of the bones; they progress from the centre toward the periphery of these organs. The bony substance often shows a fibrillary disposition; the medullar cavity is enlarged, its walls are rendered thinner; the marrow is soft and gelatinous, the adipose cells have undergone fatty degeneration and atrophy. These lesions, which are particularly marked upon the bones of the trunk and those of the upper regions of the members, vary according to the degree of intensity of the disease.

1. In the beginning and in benign cases often nothing abnormal is found on a superficial examination (Roloff); on looking closer, however, we find a hyperemia of the diseased bones, an enlargement of their channels, and a dilatation of the vessels contained therein; on section the bone is found dotted in red, the marrow is overrun with small bloody extravasations; toward the periphery of the Haversian systems the bony tissue shows but slight alterations.

2. In degrees of average intensity hyperemia is more marked; the external surface of the bone and the section show ecchymoses; the marrow is congested and overrun with hemorrhagic centres. The medullar spaces are enlarged; in the thickness of the periosteum and upon the periphery of the medullar canal we find numerous and very fine detached bony needles. The bone offers but a very slight resistance to the saw, and it may be cut with the knife; on percussion it produces a dull sound. The microscope shows a disappearance of the homogeneous structure, which is so defined in the bone normally; this is almost transparent—the osteoplasts themselves are less opaque, their volume is increased, their general form is ovoid, spherical, or polyhedral, their prolongations have disappeared; most are transformed into adipose cells. This state represents the return of the bony substance to an osteoid or medullar condition.

3. In the gravest forms, hyperemia of the bone and marrow is still more intense, the diaphyseal wall is spongy, soft, and friable; the bony substance and periosteum are partially reabsorbed; the medullar substance, on the contrary, is abundant, it pushes the bony wall aside and penetrates into the epiphyses; the bone is ruptured by the slightest pressure. When general troubles of nutrition are manifested, the marrow itself is moist, soft, and gelatiniform; it has a dirty-yellow coloration. Notwithstanding that the bone contains a smaller proportion of water, its specific weight

has diminished, a phenomenon which is due to the disappearance of calcareous salts.

Besides these lesions of the bony substance, we find at times a yellowish liquid accumulated in the articulation. The other organs are unaltered at the beginning; later, when emaciation and weakness have had time to be produced, we observe the symptoms of cachexia: hydremia, serous transudations in the large splanchnic cavities, an entire disappearance of the fat, pale, and flaccid muscles, a gelatinous infiltration of the connective tissue, etc.

Symptoms. In its initial phase osteomalacia is not marked by any appreciable symptom; emaciation, manifestations of pain and drying up of the milk secretion may only occur after quite a long time. It is very frequently preceded by slight digestive troubles and symptoms of licking-disease; but these phenomena may also be wanting. (Concerning the relations existing between these two affections, see Licking-disease, in Vol. I., page 54.)

Generally we observe only the manifestations proper to the advanced periods of osteomalacia. The gait is staggering, laborious, and painful; the lameness appears abruptly after violent exertions, long walks, etc. In the standing position the animals stamp and rest themselves first upon one leg and then upon the other; at times attacks of spasmodic contraction may be noticed upon the posterior extremities; lying down, rising, expulsion of excrements and micturition are associated with pain. The patients are very sensitive to pressures which are made upon the shoulder, the withers, and the lumbar region; during the last period of the affection they remain constantly lying down. Goats appear to be entirely paralyzed; their movements are very painful. We often find symptoms of arthritis: articular tumefactions, creaking of the joints, etc. According to Roloff, this complication is not observed on animals which are left at rest; they are said to be produced under the influence of movement: tendons and ligaments would be loosened from their periosteal attachment, and the inflammation extended to the synovial capsules. Later we notice the appearance of strains, cracks, and fractures; they may be occasioned by the slightest exertions, such as lying down, rising, turning, slipping, parturition, etc. Fractures of the ribs and pelvis are the most frequent; those involving the pelvis are usually located upon the neck of the ilium, in the neighborhood of the coxo-femoral articulation; they are at times numerous (Maris has observed as many as fifteen

fractures of the pelvis in the cow). All are almost painless, and have but a slight tendency to become cured. The observations of Roloff show, however, that they may become consolidated.

During the course of the disease, the duration of which is generally several months, emaciation is gradually accentuated, the skin becomes dry and stiff (hard skin), the appetite diminishes by degrees, weakness increases, and we note the appearance of lesions from prolonged decubitus. There is sometimes observed an impetiginous eczema (Hering). When the animals are not killed they perish from cachexia.

Prognosis. This depends mainly upon the possibility of changing the conditions in which the affected subjects are kept. In serious and much advanced cases the cure may be obtained by sending the animals to other regions, or even by a simple change of regimen. But when circumstances do not permit any useful intervention, the patients are doomed to certain death, which is not far off if fractures have already been produced. Quite frequently a spontaneous amelioration is observed when lactation has ceased, but it is only temporary; the trouble returns as soon as the females are in a gestating condition. In Norway, out of 3240 cases which were affected by osteomalacia during the year of 1877, 281 had to be killed as incurable (Krabbe).

Differential diagnosis. We may confound recent osteomalacia with articular and muscular rheumatism—affections which, moreover, complicate the former quite frequently. When it runs an enzootic course it is always easy to recognize; then, in fact, we are guided by etiological circumstances. The age of the patients permits us to differentiate it from rhachitis, which exists only in young animals. As a diagnostic symptom, Anacker mentions the clear and hollow sound which the osteomalacial bone gives on percussion, and which contrasts with the dull and full sound of the normal bone. In the goat the actinomycotic tumefactions of the bones of the head were formerly taken for osteomalacia. The localization of these alterations and their particular characters are sufficient to enable us to recognize its nature immediately.

Treatment. First of all, the causal indication must be attended to: change of regimen, a good quality of food, and food rich in calcareous salts (leguminous grain, oats, dry clover, rape cake, bean and pea grass), and in some cases a change of drinking-water. But the most efficient means is the sending of the animals

to a locality where the food contains plenty of lime salts. Through agricultural ameliorations (draining of swampy ground, manuring, etc.) we may reduce in a great proportion the losses caused by osteomalacia. With this object it is particularly advisable to use bone powder and acid calcareous phosphates as manure.

In the second place, it is proper to administer phosphate of lime in a form as assimilable as possible. Prepared bone powder is very useful; it is given in a dose of 25 to 50 grammes per day (a tablespoonful at each meal). If this preparation is not at hand we may use raw official¹ phosphate of lime; the dose is the same as that of the preceding drug. It is also recommended to give small repeated doses of phosphorus (for the ox, 0.01 to 0.03 gramme per day). Stomachics are advisable in order to influence the assimilation of nutritive principles and of the calcareous salts. Harms has pointed out the favorable effects which may be obtained by using hydrochloric acid. Roloff has also advised to milk the cows only partially, with the object of lessening the withdrawal of calcareous salts. A more efficient way would consist in excluding the animals entirely from reproduction. But, as may readily be understood, reasons of an economical kind cause these last measures to be only rarely resorted to.

B. Rhachitis.

SOFTENING OF BONES: LEG DISEASE: DWARFISHNESS.

Rhachitis is a disease of young animals. It is observed particularly in young pigs and dogs; it is less frequent in the colt and the calf, still rarer in young lions and young birds. A few cases of congenital rhachitis have been mentioned (foetal rhachitis).

Etiology. As with osteomalacia, its dominating etiological condition is the want of calcareous salts in the food. In the calf it may be occasioned by the milk of mothers which are affected by osteomalacia; in the pig, by an exclusive feeding of kitchen refuse or potatoes, which explains its frequency in this species after abundant potato crops (Utz); in the dog, by exclusive feeding of meat, when this is given without the bones. Röhl has seen it becoming

¹ The German Pharmacopoeia possesses a *calcium phosphoricum crudum* for veterinary use; this official preparation is composed of calcinated bone (ivory and stag-horn dusts); it contains tribasic phosphate and carbonate of lime, carbonate of magnesia, and fluoride of calcium.—N. D. T.

developed in young lions which were fed on boneless meat. Utz has observed it, though more rarely, in localities with a calcareous soil than in those which were mainly composed of sand, granite, and gneiss. The want of lime salts in the soil incontestably plays an etiological part.

The influence of defective breeding is aided by the food. A very stimulating alimentation, given with the object of producing or aiding precocity, permanent stabling, fattening in connection with development of the skeleton, want of air and space, are causes which favor the development of rhachitis, and which also explain its rarity in pigs living in fields. Some authors have attributed the disease to cold, damp stables, cold floors, and draughts (Stockfleth); but the action of these causes is entirely secondary. It is possible that gastro-intestinal catarrhs, in the course of which the absorption of calcareous salts contained in the food is lessened, favor its development. Certain much improved breeds of pigs seem to possess a congenital predisposition to it; since the importation of English breeds it has become much more frequent in young pigs. If any infectious matters interfere with the genesis of rhachitis, their nature and mode of action are entirely unknown.

[The too great simplicity of the food, or insufficient variety—when associated with certain conditions, especially want of exercise in the open air, bad sanitary conditions, depending upon improper ventilation and drainage; together with an inadequate supply of natural light (sunlight)—is one of the predominating causes of this disease in dogs, in menagerie animals, and sometimes also in horses and other animals.—W. L. Z.]

Pathological anatomy. The want of solidity of the bones, due to an insufficiency of lime salts, is the fundamental alteration of rhachitis. It is always accompanied by particular and very remarkable vegetations of the periosteum as well as the epiphyseal cartilages.

1. The periosteum is hyperemic and infiltrated; its internal face is doubled by a new layer; the osteogenetic layer is thickened; the morbid tissue remains soft (spongoid layer), later it undergoes a certain degree of calcification; the bone, the volume and weight of which are increased, are covered with exostoses; these periosteal proliferations are especially found at the point of attachment of the muscles. In the pig they are usually much developed upon the

femur, upon the points of insertion of the great psoas, of the iliac psoas, and upon the head of the calcaneum. The muscular contraction may remove the thickened periosteum; this lesion is quite common upon the scapula (in the pig).

2. Alterations which are more marked exist upon the epiphyses; we find an abundant proliferation of the epiphyseal cartilage in it, or insufficient calcification. In a normal state the cartilage which is situated between the epiphysis and diaphysis is separated from the latter by two thin parallel layers: the proliferating and the ossifying layer; in the rhachitic bone the first is extremely developed, while the second is hardly perceptible; moreover, these layers are no longer parallel, they become serrated and penetrate into each other. In the cartilage which is undergoing proliferation we find scattered centres of medullar substance and others in course of calcification. Upon the surface of the ossified layer a very vascular spongy tissue is found. The exuberance of these morbid tissues produces incurvation of the diaphysis, a thickening, a tumefaction of the epiphyses and sometimes their loosening, which is favored by a less close union of these parts with the diaphysis.

This abnormal proliferation of the cartilage and of the periosteum produces various deformities. The bones have been arrested in their development (dwarfishness); they are reduced, thick, heavy, and *knotted*, especially in the neighborhood of the articulations (double articulations); they are frequently incurved. The members are the seat of deformities which have been the cause of the terms *badger* or *sword legs*. The spinal cord is embossed in certain points and depressed in others; it is at times incurved in the largest part of its length, its upward incurvation being designated *kyphosis* (carp back), its downward incurvation *lordosis* (saddle back), the lateral derivations are called *scolioses*. Bony vegetations are also observed upon sternum (chicken breast), pelvis, etc. Some diseased bones form an elbow, others become fractured. In young rhachitic dogs, Schütz found fontanelles upon the cranial bones. Secondly, we may note the appearance of articular lesions; softening and malleability of the bones favor traction and distention of the ligaments, and arthritic complications are frequent in rhachitis. The articulations of the ribs with their cartilaginous prolongations are often affected; the tumefactions of which they are the seat constitute a *rhachitic rosary*.

Symptoms. The evolution of rhachitis is always slow, and

often its symptoms are only much marked after several months. We will review these symptoms in our different species.

1. In the PIG the disease is sometimes announced by symptoms of licking-disease, at others by weakness, indolence, and stiffness of the gait (*stiff* young pigs). The back is arched, the diseased legs are the seat of spasmodic contractions, the animals remain almost constantly down. The epiphyses and the sternal prolongations of the ribs are tumefied and painful. The bony rays become deviated and *knotted*; the feet become incurved forward, backward, in- or outward; the astragalus often touches the ground; strong pressure exerted upon the diseased bones produces at times a cracking noise. We may observe *kyphosis*, *lordosis*, and *scoliosis*; the pelvis becomes depressed upon the surface of the coxo-femoral articulations, a deformity which may offer mechanical obstruction to parturition (*rhachitic pelvis*). Sometimes we observe upon the jaws and bones of the nose a swelling which renders mastication and breathing difficult or impossible (Lafosse); this state represents a form of the *disease of sniffing* (see the Addenda). Deformities of the bones are accompanied by arthritis and articular tumefactions (hock-joint, knee, or fetlock). The development of the animals is obstructed, dentition is also delayed, the appetite is diminished, and later it disappears. The patients often remain *dwarfy*, they are emaciated, are affected by diarrhea, and remain almost constantly lying down. Rehms has seen cutaneous impetiginous eruptions, and Lafosse bronchial catarrh. Death follows from the progress of cachexia.

2. In the DOG rhachitis is also marked by bony tumefactions, which, as a rule, are very evident upon the articulations of the ribs with their cartilaginous prolongations (*rhachitic rosary*), by a deformity of the extremities (*badger* or *sword legs*); the radius and cubitus are incurved forward; the walk is stiff and laborious; the support is extended to the whole region of the toes, the calcaneum often touches the ground; the epiphyses become tumefied (*double articulations*); the *chicken breast* becomes gradually formed (the vertebro-sternal diameter increases while the bi-costal diameter diminishes). Dentition is often delayed; the teeth are small and the enamel rugous. Cutaneous eruptions are frequent.

3. In the HORSE the development of the organism is delayed; the slightest efforts are accompanied by fatigue and shortness of breath. The tumefactions appear ordinarily upon the epiphyses

of the legs, mainly upon the tibia, and bones of the head; upon these latter they seem to be occasioned by pressures of the halter; those of the maxillaries, which are not rare, appear to be due to the eruption of the molar teeth, which are always late. When these alterations are accompanied by contractions of the nasal cavities and chronic catarrh of the pituitary they obstruct respiration (Hörner). Incurvation of the extremities is generally less accentuated in the pig and dog; we may, however, find some deformities of the forelegs which become similar to the twisted legs of the dachshund. Often also the rhachitic colts are knuckled and bow-jointed in the front members (*bear feet*); we observe as a rule lameness, articular tumefactions of the hock (*pseudo-spavin*) and knee joint; the altered bones are predisposed to fractures. Some animals have a saddle back, others have a carp or roach back. At certain times the disease produces pains, which the patients manifest by scratching the ground, stamping, also by contracting the hind legs spasmodically or by groaning. It predisposes the colt to eczematous cutaneous eruptions, to bronchial and intestinal catarrh, to lesions of the thyroid gland, and to accidents the result of prolonged decubitus.

Bran disease is but a form of rhachitis (see Addenda). Of all young animals the colt is the one which enjoys the best care, the most rational alimentation, and the best regulated hygiene. It is unnecessary to seek elsewhere for a cause of the scarcity of cases of rhachitis which are found in these animals.

4. In the OX the ordinary symptoms of rhachitis are tumefactions of the bones of the carpus and tarsus, incurvation of the back and extremities (X-legs and crooked hocks), the rhachitic rosary and pelvis, a laborious, painful walk, and a permanent decubitus.

5. In GALLINACEÆ rhachitis is not equally frequent in all the species; it is quite common in chickens which are three to six months old (Zürn); it is rarer in pigeons, geese, and ducks. The affected birds remain seated continually, the walk is laborious and stiff; the articulations of the extremities (feet, wings) are *knotted*; the bones are soft, malleable, incurved, and the breast-bone is deformed. The affection speedily leads to emaciation and enemia.

Prognosis. It is relatively favorable when it is possible to suppress the causes of the trouble. Much accentuated bony neoformations may disappear entirely in young subjects, but the de-

formities persist. It is economical to sacrifice the subjects early, when their meat may be used for consumption.

Differential diagnosis. Rhachitis may be confounded with pyemic arthritis and acute articular rheumatism. These two diseases are recognized by their sudden invasion, their rapid evolution, by the intense febrile reaction by which they are accompanied, and lastly by the absence of tumefaction and incurvation of the bones, alterations which belong to rhachitis. Pyemic arthritis is also distinctly characterized by abscess formation of the joints.

Treatment. The treatment of rhachitis comprises the same indications as that of osteomalacia. The regimen should be changed, and selected food must be given; alimentary overloading of the stomach must be avoided, and gastro-intestinal catarrh must be combated if requisite; lastly, new breeding stock is to be used. Rhachitic pigs must be driven to the field and subjected to the vivifying influence of the open air. Prepared bone dust must be given in a dose amounting to a tea- or tablespoonful, according to the size of the animals; in the dog we may give official phosphate of lime (5 to 10 grammes per day).¹ Natural phosphorus when administered for a sufficient time accelerates ossification; in large-sized colts it is given in a dose of 0.01 to 0.05 gramme dissolved in ordinary oil or cod-liver oil; for the dog, in a dose of 0.0005 to 0.002 gramme. For the horse we prescribe: phosphorus 0.05; cod-liver oil 300 grammes; this solution is to be administered in a bran slop; for the dog and pig, phosphorus 0.03 gramme; cod-liver oil 300 grammes; a tablespoonful per day. For poultry Zörn recommends grains of lime phosphate and meat pills. The inhabitants of the neighborhood of Lake Ammer treat with success their rhachitic chickens by feeding them with fish. The calcareous skeleton of fish acts in concert with nitrogenous food.²

ADDENDA. I. The disease of the horse which has been described under the name of **BRAN DISEASE** is but a form of rhachitis. It appears to be produced by an exclusive alimentation with bran, and is observed most frequently in millers' horses. Its first manifestations are digestive troubles, constipation, weakness, fatigue,

¹ A therapeutic agent generally used nowadays is chlorhydro-phosphate of lime. Ten centimetre cubes of this product contain a gramme of tricalcic phosphate and one centimetre cube of hydrochloric acid. At the time of its administration it should be diluted with five times its weight of water.—N. D. T.

² Mertz: (Unpublished communication.)

and very abundant perspiration under the influence of the slightest exertion. Soon we notice the appearance, in the neighborhood of the articulations of the knee and tarsus, bony tumefactions accompanied by lameness and painful spells; these alterations appear also upon the bones of the head, especially upon the jaws and in the bones of the nose; prehension and deglutition are difficult or impossible, the teeth loosen and fall out. The animals become weaker and weaker, and die in a cachectic condition. It is impossible to differentiate these symptoms from those of rhachitis.

Bran is just as improper a food for raising the colt as is potato for the young pig; both are very poor in calcareous salts. Bran given as exclusive food may thus lead to the same consequences as the food mentioned in the article upon the etiology of rhachitis. It produces also a harmful action which is due to the superabundance of phosphates which it contains. According to this Pütz has assimilated *bran disease* to chronic phosphoric poisoning, and has compared it to phosphorus necrosis of the maxillary of man. This hypothesis is not well founded, for free phosphorus alone, and not phosphoric acid or phosphates, possesses an irritating action upon the bony tissue. In old animals, *bran disease* is identical with osteomalacia. Have not some cases of actinomycosis been ascribed to this affection?

II. Under the typical denomination of SNIFFING DISEASE several authors have described, in the pig, a protean disease which sometimes seems to be rheumatism, at other times a chronic hemorrhagic, or purulent catarrh of the nose, and, lastly, often tuberculosis or actinomycosis.

On account of this complexity, SNIFFING DISEASE could not be classified in a nosologic list.

The symptom which is common to the affections which the ancients have confounded under this appellation is the *wheezing respiration (sniffing)*, which is intermittent in the beginning, but permanent within a certain time; it is the consequence of the tumefaction of the upper maxillary, of thickening of the nose, shortening and incurvation of the groin.

a. In the *catarrhal form*, we observe, in the beginning, a seromucous discharge which becomes successively hemorrhagic and purulent; epistaxis is common, facilitating respiration momentarily; besides these phenomena, we find conjunctivitis and a more or less frequent cough; the prehension of food is difficult, there are

also nauseas and emaciation ; finally death occurs through cachexia or asphyxia. At the autopsy we find a hemorrhagic and purulent inflammation of the nasal mucous membrane, incurvation, atrophy of the turbinated and ethmoid bones. Schneider maintains that the cause of this catarrhal form of the affection resides in the congenital rudimentary development of the turbinated and ethmoid bones, which would favor the accumulation of foreign bodies in the nose and the development of rhinitis.

b. The *rhachitic form*, the one which it is necessary to mention here, coexists with the special alterations of the extremities. Haubold, who has observed several of these cases, has always found arthritis. Lafosse has also observed all the symptoms of rhachitis. In young horses, during the course of rhachitis, we observe an entirely similar tumefaction of the bones of the head, especially of the walls of the nasal cavities, and also an obstructed respiration.

At the present time we do not possess any positive fact upon the other varieties of *sniffing disease*.

The *treatment* comprises different indications according to the form of the affection, but we must not rely upon it. It is always better to kill the patients.

III. Under the denomination of OSTEOPOROSIS we designate, in pathological anatomy, a dilatation of the Haversian canals, with atrophy or absorption of the compact bony substance. Osteoporosis may be observed in the course of osteomalacia. Notwithstanding Dieckerhoff, it must not be considered as a special nosological species.

In cases where bony absorption is produced by medullar changes, the atrophy resulting from it is called *excentric*; when it proceeds from the surface it is qualified as *concentric*. These two forms of the atrophic process which are observed in osteomalacia do not constitute any special affections any more than osteoporosis. There is besides an intimate affinity between these morbid conditions.

IV. ACUTE INFECTIOUS OSTEOMYELITIS of man is a pyretic, microbic disease, which is frequent in Southern Germany, Switzerland, and on the coasts of Northern Germany. It is a disease of young animals. It is characterized by purulent or gangrenous inflammatory centres which are developed in the marrow of long bones (femur, tibia, more rarely in the radius and cubitus). It is also designated under the names *primitive osteomyelitis*, in order to

differentiate it from *secondary osteomyelitis*, which appears in the course of other infectious diseases (typhus, scarlatina, measles, pyemia, tuberculosis, cystitis, etc.), or in poisonings.

Primitive infectious osteomyelitis is determined by special microbes (*Staphylococcus pyogenes aureus* and *albus*).

Stickler¹ has described alterations of the marrow of the bone in some diseases of the horse, but we do not as yet possess any precise information upon the existence, in our animals, of primitive infectious osteomyelitis.

TRICHINOSIS OF THE PIG.

NATURAL HISTORY. The *Trichina* (*Trichina spiralis*) is a small filiform nematode worm (nematelminth) of the Tricho-trachelides family. It was introduced into Europe about 1830, by Chinese pigs; it spread very rapidly. Owen described it in 1835, and gave it the name which it now bears. Leidy found it in the pig in 1847, and Zencker in man in 1860, in Dresden. It appears under two forms, which are very different as far as its stages of evolution are concerned. We distinguish sexuated *intestinal trichina* and asexuated *muscular trichina*.

1. *Intestinal Trichina* is only found in the intestine; it is straight, and provided with a pointed head; the length of the male is $1\frac{1}{2}$ millimetres, that of the female from 3 to 4 millimetres. The digestive apparatus comprises a buccal opening, œsophagus, stomach, intestine, anus, and cloacal slit. The genital organs are formed in the male by the testicular tube, the deferent canal, and the genital orifice; in the female, by the ovary, uterus, vagina, and vulva. The duration of life of this parasite is from five to six weeks; during this time one female alone gives birth to nearly 1500 young ones.

2. *Muscular Trichina* represents a larval form of intestinal trichina. It reaches a length of one millimetre; the front part of its body is pointed, the hind extremities are rounded and split. In the muscular substance it exhibits the formation of rounded or ovoid capsules containing from one to four parasites, which are twisted upon themselves in various ways. In this state it may preserve its vitality for a long time; for eleven years in the pig (Dammann), for twenty-four years and more in man. Our pres-

¹ Stickler: Berlin. Archiv, 1887.

ent knowledge of the development of trichina is especially due to the researches of Leuckart, Zencker, Pagenstecker, Haubner, Virchow, etc. The infection is produced by meat containing trichinæ, but we do not as yet know if the embryos ejected with the excrements are taken back directly. It is possible that non-digested pieces of meat infested by trichina may be contained in the excrements; in these conditions these may infect healthy animals.

The developments of the trichina offer four successive phases for our consideration :

1. The period of formation of the embryo in the intestine. It begins toward the seventh day after the ingestion of the meat. Encysted muscular trichinæ become free within twenty-four hours; the capsule which contains them is dissolved by the gastric juice. Twenty-four to forty-eight hours after becoming free the parasites, which reproduce readily, copulate; a few days later living embryos exist in the intestine.

2. The emigrating period, which is prolonged till the second or third week. From the seventh day the embryos begin to perforate the intestinal walls, and, passing into the connective tissue, reach the muscles. The duration of this migration is from nine to ten days.

3. The encysting period in the muscles. This extends from the fourth week to the third month. After reaching the muscular tissue the embryos seem to sleep for about two weeks, then they become transformed into larval trichinæ. The perimysium is the seat of an abundant cellular infiltration; the capillaries are much dilated; the muscular fibres lose their transverse striation, the sarcolemma is dilated and spindle-shaped around the parasites which are within, its glands become proliferated, and the primitive fibres disappear. Gradually cellular layers become regularly disposed around the trichina in order to constitute an oval, fusiform capsule having two opaque poles on the extremities. This is the time when the danger of contamination through meat begins.

4. The period of calcification of the capsule. It may last from the third month to one and a half years. This calcification hides the trichina; but in very thin sections it is possible, by a careful examination with the naked eye only to observe small, calcareous islands which contain them; they are about one millimetre in length, and appear under the form of clear spots. Sooner or later the trichinæ themselves become calcified or undergo fatty degenera-

tion. Outside of the muscles we find also trichinæ in the adipose tissue (bacon), sometimes in a free state, at other times encysted (Chatin).

AFFECTED ANIMALS. Trichina has been observed in man, also in the pig, rat, mouse, dog, fox, bear, polecat, marten, the domestic fowl, turkey, jay, and in some fish, especially the pike. Experimental transmission has been successful in the horse, ox, sheep, rabbit, and guinea-pig. The selected organs of the embryo are the pillars of the diaphragm, tongue, masseters, the cervical and thoracic muscles, and the muscles of the thigh. The adipose tissue in the neighborhood of the muscles shows them frequently. The number of trichinæ contained in the different muscles is extremely variable; we may find as many as 1500 in one gramme of meat.

The statistical researches which have been made upon the frequency and dissemination of trichinosis of the pig have shown that this disease is much more common in Northern Germany than in the southern provinces. Out of 20,000,000 pigs examined in the Kingdom of Prussia, from 1876 to 1882, we found 10,000 affected by trichinosis; in Prussia there is 1 affected pig in about 2000; an average proportion for the whole German Empire is 1 per 10,000 ($\frac{1}{10}$ per 1000). In America this proportion is much greater; according to the researches by Billings, of Washington, it is said to be 4 per 1000. The examinations which have been made in Germany upon pork imported from America have given an average of 21 to 30 per 1000 (maximum, 80 per 1000).

Pathology. The mode of infection in the pig is as yet known imperfectly. It is generally admitted that rats and mice are its agents. Rats affected by trichinosis abound in pens and slaughter-houses: of eighteen pens visited by Leisering, fourteen contained rats so affected. In some rats (77) coming also from the skinning enclosure, Franck observed a proportion of 9 per cent. affected by trichinosis. Upon 704 rats of various origin, Heller found 8 per cent. to be thus affected.

But if the pig usually contracts trichinosis by eating rats and mice, he may also become directly infected by eating either some remains of trichinosed pork or waste from the meat market, also excrements of animals belonging to his own species containing non-digested trichinosed meat. The observations of Hertwig seem to demonstrate that the invasion of the parasites may take place in several ways; this author found in the muscles trichinæ in various

stages of development. Nothing precise is known upon the possibility of an infection by the embryos of intestinal trichinæ which are rejected with the excrements. As for the existence of congenital trichinosis, it is problematic.

Symptoms. We have never observed symptoms of purely accidental trichinosis of the pig, but those of experimental trichinosis have been studied. The first, which are ephemeral and hardly evident, pass unobserved; they could only be found during the period of the invasion of the parasites, and the owners do not observe it, or they mistake its nature; later, when the invading period is over, the general conditions of trichinosed animals is not at all affected. Thus, usually at the time of slaughtering the period of the invasion of the trichina has long since passed, and we may thus explain the absence of morbid phenomena in the last period of the life of animals which are found much infested at the slaughterhouse. Small numbers of trichinæ do not show any manifestation.

The symptoms of experimental trichinosis are related to an *intestinal affection* or to *muscular lesions*.

1. The *affection of the intestinal canal* becomes well marked toward the end of the first or the beginning of the second week after infection by a lessening of the appetite, depression, arching of the back, tension of the abdomen, an obstinate diarrhea preceded or accompanied by colics, by weakness and exhaustion; in some cases we observe vomiting. In very young animals and in those in which trichinæ exist in large numbers in the intestine, the disease often leads to rapid death. When the parasites are not very numerous it may stop in this first period.

2. The *symptoms* produced by muscular lesions appear as soon as the trichinæ which have penetrated into the muscles determine inflammatory symptoms therein; they are observed from the second to the third week. The animals are tormented by a very singular pruritus; they rub and scratch themselves constantly; the extremities are stiff, and this state may become transformed into true paresis; the patients suffer and lose flesh rapidly. We observe also difficulty in swallowing and mastication (trismus); respiration is laborious and painful, the voice is hoarse and weak; sometimes oedematous tumefactions appear in various regions. Generally a complete cure is produced within four to six weeks. The animals recover their strength and condition gradually; death is an exceptional ending of this trouble.

Diagnosis. In the majority of cases it is impossible to recognize trichinosis during life. Its symptoms, which are always very vague, offer too much analogy with those of intestinal catarrh or muscular rheumatism to render it possible to make a distinction, and when once the trichinæ are encysted, their presence is not betrayed by any important symptom. The search for parasites in the meat is easy. When the cysts have undergone calcareous infiltration, they are marked, and found, on section, in the shape of small white dots, distinguishable by the naked eye. But the diagnosis is only rendered certain by microscopic examination. Meat inspectors generally use a magnifying glass of ten diameters; they used formerly a glass of twenty-five to fifty diameters; this practice has been abandoned because it requires more time than the former, which answers perfectly well. The technique of the search for trichinæ is very simple: with small bent scissors we make in the muscular tissue, in the neighborhood of the bones, sections (as thin as possible) which follow the direction of the fibres; these sections must be compressed between two glass slides to the extent of rendering them transparent, and then they are carried upon the stage of the microscope. In order to be sure of the result it is the rule to examine muscular fragments coming from six different regions (diaphragm, eye, larynx, intercostal muscles, neck, and tongue), and to make six preparations with each fragment. In Prussia a recent order prescribes the making of half of the preparations with muscular tissue obtained from the pillars of the diaphragm, where the trichinæ are particularly numerous.

The possible presence of trichinæ in the adipose tissue renders the examination of bacon and lard necessary (Chatin).

Differential diagnosis. Encysted trichinæ may be confounded with other muscular neoformations. Among these latter, we must specially mention *concretions*. They are whitish-gray corpuscles, of the size of a millet-seed, the muscles of which are sometimes invaded. Their nature is very varied; sometimes they are formed by lime crystals, tyrosin, margarin, and stearin, at other times by calcified cysticerci or miliary neoplasm. Formerly they were taken for guanin concretions. They are altogether different from cysts of trichinosis by their dimensions, their form, and by the absence of nemathelminths within them; besides, they possess neither the characteristic form, "eye-shaped," of trichinæ, nor their dark poles or the clear definition of their contours.

We may mistake trichinæ for Miescher's tubes ; but these latter are deprived of capsules. They are elongated, blunt at their extremities, and strangulated into graded pouches ; they are of larger dimensions than those of the trichinæ, and are formed by a dark granulous mass, which, under a greater enlargement, is shown to consist of a quantity of halfmoon-shaped corpuscles ; lastly, the transverse striation of the muscular fibres of the neighborhood of Miescher's tubes is perfectly preserved.

It is easy to differentiate trichinæ from actinomycetes ; the latter are fungi which appear under the aspect of rounded disks or in the form of beans, and are ray-shaped, with a thin centre and thick edges. When crushed, they show themselves to consist of filaments which are enlarged in the shape of a club upon their free extremity, which is very refringent. Other helminths, such as the distomas, which are located in the muscles of the larynx and pillars of the diaphragm, may be taken for trichinæ at first sight, but the two cupping orifices with which the distomas are provided, their cylindrical form, and their very active movements, are characters which enable us to recognize them. Measly spots, the echinococcus, rhabdites, etc., must also be mentioned in connection with the differential diagnosis of trichinosis.

Ebertz¹ found in the sheep a parasite of the muscles which is quite similar to trichiua. This parasite, which has been found in several preparations of the muscular substance, was smaller than the trichina, and performed very active undulatory movements. According to Leuckart, it would represent the larval form of a hair-worm or strongylus.

Prophylaxis. It is useless to spend any time upon the treatment of trichinosis ; it is impossible to kill the parasites in the affected animal.

Among the prophylactic measures we must especially mention ; complete destruction of the cadavers of trichinosed pigs (cremation) ; very attentive examination of meat from the markets ; the destruction of rats and mice in pig-pens, prohibition of the raising of pigs in slaughter-houses and curing-rooms, finally, the exclusion of meat in the alimentation of this animal.

TRICHINOSIS OF MAN. Since trichinosis was known (1860) thirty epidemics of it have been observed ; those of Hettstädt

¹ Ebertz: Eulenberg's Vierteljahrsschr., Bd. xlvii.

(1863-64), Hedersleben (1865), and Niederzwehren (1877), have been particularly serious.

Its manifestations are variable. During the first eight days we observe symptoms of gastro-intestinal catarrh (inappetence, uneasiness, vomiting, diarrhea, fever, colics, and choleric accidents); from the tenth day muscular symptoms dominate the scene (muscular pain when moving, *tetanic* tumefaction and hardness of the diseased muscles, flexion of the articulation during decubitus, difficulty of mastication and dysphagia, hoarse voice, aphonia, dyspnoea, oedema of the eyelids, the face, and the extremities, fever, etc.). The treatments which have been tried (purgatives, benzine, san-tonin, essence of turpentine, glycerin, etc.), have not given any beneficial results.

Contamination is always produced by the use of raw or incompletely cooked pork. Prophylaxis should exclude these meats from alimentation. Boiling and roasting may, however, render trichinosed meat harmless: at a temperature of 62° - 75° C. albumin becomes coagulated and the trichinæ perish, but cooking must be prolonged for quite a long time. The researches of Vallin have shown that a piece of beef weighing three kilos reaches, in its centre, a temperature of 90° - 100° C. after four hours' boiling only; in the first hour the central temperature reaches but 50° C. A boiling of three hours is not sufficient to kill all the trichinæ in a ham. The preparation of roast beef needing but a temperature of 50° to 60° C., and a leg of mutton 48° to 56° C., these culinary processes could not protect us from infection. Trichinæ are destroyed by thorough salting acting for several weeks, and by hot smoking lasting for eight days at least. A pressure of twelve atmospheres kills them in a relatively short time. The researches of H. Bouley and Gibier seem to establish that meat becomes inoffensive when under the influence of prolonged cold. The experiments of Lenckart have, however, given contrary results.

Microscopic examination of meat offers assuredly less guarantee than the prophylactic means of which we have just spoken.

CYSTICERCOSIS OF DOMESTIC ANIMALS.

I. Measles of the Pig.

NATURAL HISTORY. Formerly known as *granulous disease*, *pearled disease*, measles of the pig is a parasitic disease caused by

the *Cysticercus cellulosæ*, a larval form of the *Tænia solium* of man. Küchenmeister and Haubner enjoy the credit of having demonstrated, by the method of experimental infections, the relation existing between teniasis of man and measles of the pig.

The frequency of measles corresponds with that of tapeworm. The pig becomes infected by eating human excrements which contain proglottides of *Tænia solium*. The avidity of this animal for human excrements predisposes it to measles. The danger of infection is so much the greater when the animals can find human excrements easily (when being driven into the fields; also in unclean villages, having no privies; also when privies and pig-styes are in proximity). The mode of raising and keeping the pig has, therefore, a great influence upon the frequency of the disease.

After having reached the stomach the eggs of the *Tænia solium* are freed from their shell by the dissolving action of the gastric juice. The hexagonal embryo becomes free; at times it perforates the intestinal wall and progresses into the connective tissue, at other times it is washed out by the blood current. These migrations take place only in the young pig not over six months old; adult subjects do not become infected. When the embryo is fixed upon some point it loses its hooks and becomes translucent in its centre; the elements of the tissue which surround it form a vesicle upon the inner surface, from which a papilliform body is developed—the base of the *scolex*. Within twenty days the embryo will have reached the dimensions of a pin's head, and the future head is marked by a dark spot; toward the fortieth day the cysticercus reaches the size of a mustard seed; the head is then very distinct, the cupping apparatus and crown of hooks are yet rudimentary; at the end of two months the grain of measles, of the size of a pea, the crown of hooks, and the cupping apparatus are plainly visible, but the neck is wanting; after the third month, finally, the cysticercus is entirely developed and ready to infect man. The tissues surrounding the parasite become the seat of an inflammatory reaction, the result of which is the production of a connective tissue capsule.

The grains of measles are usually found in the interstitial connective tissue of the muscles of the tongue, heart, neck, and shoulders, pelvis, flank, and superior regions of the legs, also in the subcutaneous connective tissue, in the brain and in the spinal cord, in the eye, the liver, spleen, lungs, splanchnic cavities, etc.;

it exists sometimes in bacon. Quite frequently all the organs are overrun by it; they may be found by thousands, two or three in a single gramme of meat; infrequent and isolated cysticerci are much more rarely observed.

Adult cysticerci are of the size of a pea or bean. They represent vesicles which are of a dull white color, of soft consistence, and provided with a head and neck. The first, which is marked externally by a dark spot, is bent in toward the inside of the vesicle, a disposition which renders this reniform; when it is grooved the vesicle has the aspect of a bottle. The head is provided with four cupping apparatus, a rostrum, and a double crown of twenty-six to thirty hooks. The vesicles are generally imprisoned in a capsule of the connective tissue (except those developed in the serous cavities); they creak under the cutting instrument, and burst when the meat undergoes boiling; the dead vesicles undergo fatty degeneration and become calcified. The neighboring muscular tissue is intact at the start; later, when the cysticerci exist in large numbers, the muscles become viscous and infiltrated with serum.

The frequency of measles in the pig varies with the regions. In Prussia, out of 1000 pigs, an average of three are found to be affected by the disease (statistics from 1876 to 1882, on 20,000,000 animals).

Symptoms. They have nothing very marked; many animals much affected by measles do not show any morbid manifestation during life. In a general way we consider as symptoms of measles a hoarse voice, falling out of the hair, depression, general weakness, inappetence, paleness of the buccal mucous membrane, anemia, emaciation, œdema of the head, neck, and shoulders, etc., diarrhea, general paresis, or local paralysis.

Independently of these general and vague troubles, others are observed more precise, and due to the presence of cysticercus in the different organs; such are cerebral accidents, epileptiform cramps, hemiplegia or general paralysis, and rabiform symptoms of an encephalic localization; blindness at the time of the migration of the cysticercus into the eye (under the retina, in the crystalline lens, and the eye chambers); paralysis of the tongue and incapacity of prehension when the measly vesicles are numerous on the tongue; peritonitis and pleurisy when the parasites become localized upon the serous membranes (Engel). Within a variable time death occurs by exhaustion. In certain cases—when, for instance, the eggs of

the tænia are ingested in very large numbers—it is produced very rapidly. We have seen cases of sudden death.

Diagnosis. A diagnosis *intra vitam* is only possible when the cysticerci are apparent on the lower side of the tongue, in front or on the sides of the frenum, or on the internal surface of the eyelids; they constitute small pimples, which may be felt or seen through the mucous membrane. In cases of legitimate suspicion, Gerlach recommends to make a longitudinal incision in the thickness of the tongue, and thus to uncover the measly vesicles. Perhaps in the future the ophthalmoscope will render us service in the diagnosis of measles. The absence of cysticerci on the lower surface of the tongue does not at all warrant us in concluding the non-existence of measles. Out of forty-one pigs affected by measles examined by Railliet, ten had the tongue in a healthy condition.

The recognition of measly vesicles upon the dead animal is very simple, except in cases where the cysticerci are dead or obliterated by different degenerations. A diagnosis is assured by microscopic recognition of the head, the hooks, or the cystic vesicles. In sausage and hacked meat the search for the parasite requires much time and a great deal of attention. Schmidt-Mülheim advises putting the suspected substances in a glass for several hours at a temperature of 40° C., and under the action of six to eight times their weight of artificial gastric juice; by proceeding thus the entire substance is soon dissolved, except the heads and hooks of the cysticerci, which are deposited at the bottom of the glass, and which may be easily examined with the microscope. The artificial gastric juice is prepared by macerating in glycerin for a sufficient time fragments of gastric mucous membrane of the pig, and mixing with it a solution of hydrochloric acid at $\frac{1}{2}$ per cent.; glycerin pepsin is obtained in this way.

Prophylaxis. We may avoid infection in the pig by preventing it from ingesting the fecal matters of man, by keeping the pig-pens very clean, and finally by instituting a regimen of permanent stabling—measures which are especially important for young animals (less than six months old). It will be also advisable to combat the tapeworm of man and to effect its expulsion and destruction.

No treatment will check measles in the pig.

In most countries measles in the pig is recorded among the

number of vices which annul a contract of sale. The delay allowed by law to institute proceedings is eight days in Prussia, Bavaria, Hesse, Austria, Waldeck; twenty-eight days in Würtemberg, the Duchy of Baden and Hohenzollern, and thirty days in Saxony.

INFECTION OF MAN is the consequence of ingestion of meat from a pig affected by measles, this meat being eaten raw or rare; sausages are especially dangerous. For prophylaxis it is imperative to subject pork to sufficient cooking (boiling or roasting). The experiments of Perroncito have taught that the *cysticercus* succumbs at a temperature of 50° C. lasting over a minute. These parasites have perished in well-smoked meat, also in salted or dried meat; by losing their moisture they become reduced to small and hard masses, of the size of a pin's head, and which crack under the teeth.

The measures of the sanitary police relative to the consumption of meat coming from pigs infected by measles are more or less stringent in different countries and localities, but there is one which should be inexorable: it is the exclusion from consumption of meat which is overrun by this trouble. Such meat is destroyed or used for industrial purposes (for the manufacture of soap and gelatin). The same ought to be the case with bacon, which is frequently overrun with *cysticerci* which are difficult to discover; this food is so much the more dangerous from the fact that the habit of eating it raw is very common. On the other hand, meats which contain but a few rare *cysticerci* may be consumed if some precautions are taken; an excellent practice is to subject it to cooking before selling it, and to give it a special mark.

The use of measly meat exposes us to intestinal helminthiasis (tapeworm), which produces digestive and nutritive troubles, also to encephalic troubles and to auto-infection by the eggs of the *Tenia solium*. When these eggs are hatched in the human intestine the embryo may pierce the intestinal wall, and *cysticerci* are developed in the organs, in the eye, brain, etc.; we then observe cerebral troubles, blindness, and sometimes death.

II. Measles of the Ox.

NATURAL HISTORY. The *cysticercus* of the ox represents the asexual larval form of the *Tenia mediocanellata* (*saginata*, *inermis*) of man. The affection which it determines is less frequent than

measles of the pig, but it is more common than is generally admitted. It is developed after the ingestion of the proglottides and eggs which are mixed with human excrements. The cysticerci are especially localized in the muscles, heart, tongue, and adipose tissue; they are more rarely met with in the liver, lungs, brain, kidneys, etc.; ordinarily they are found isolated and in much smaller number than the *Cysticercus cellulosæ*. Hering has counted as many as 300 in half a pound of beef.

The development of the cysticercus of the ox is ideutical with that of measles; the vesicles themselves differ very little from a microscopic point of view. (Statements given as to the dimensions of the latter vary exceedingly; according to some authors they are smaller than measles of the pig; others give them double the proportions.) From a microscopic point of view the parasite is characterized by absence of the rostrum and double crown of hooks, from which it receives the name of *Tænia inermis*. The four cupping apparatus are much developed; upon the upper part of the head we see also a frontal sucker.

Symptoms. Measles of the ox is not marked by any symptom which enables us to suspect it. Produced by experiment, it may develop without occasioning any morbid symptom (Gerlach, Peroncito, Pütz). Experimental measles of the calf, on the contrary, is marked by very particular trouble, which, with its post-mortem lesions, has been the cause of this disease having been improperly called *acute cestoidæan tuberculosis*—Leuckart, Möller, Zürn). According to Zürn, its principal symptoms are a rise of temperature, inappetence, pain produced on abdomiual palpation, complaints, a staggering, stiff, and painful gait, a permanent decubitus, inability to rise, dyspnœa, diarrhea, a lowering of temperature, weakness and exhaustion. Death occurs, as a rule, within about three weeks. At the autopsy we find thousands of cysticerci in the heart, in the muscles of the cheeks, tongue, neck and shoulders, abdomen, and in the diaphragm.

Diagnosis. With the exception of cases where the parasites are apparent on the lower side of the tongue, it is absolutely impossible to recognize measles of the ox during life. According to Masse and Pourquier, in the south of France, the slaughter-house meat inspectors are said to examine the tongues of oxen in the manner usual in the case of the pig when measles is suspected.

In the cadaver, the recognition of cysticerci is much more diffi-

cult than in the pig; they have often passed unobserved. We cannot otherwise explain the rarity of measles in the ox, especially if we compare it with the frequency of the *Tenia inermis* of man. The discovery of the parasites necessitates a minute examination, for they are usually disseminated and located in the adipose tissue. Recent researches made in the Berlin slaughter-houses have demonstrated that the internal masseter (pterygoideus) constitute their favorite abode.

Prophylaxis. This bears the same indications as that of measles of the pig; but it is still more difficult to interfere beneficially in the ox than in the former animal. We must especially prevent the men having charge from depositing any excrementitious matters in the stables.

MAN is infected through the ingestion of raw or incompletely cooked beef (rare beef-steak). Thorough boiling and roasting of meat are the best prophylactic measures. *Tenia inermis* is much more frequent in some countries than tapeworm, and it is harder to expel; in man, however, it never produces auto-infection.

III. Measles of the Dog.

Cysticercus cellulosæ has been found in the brain, muscles, liver, and lungs of the dog. The disease appears to be of human origin.

In most cases we have observed symptoms of a cerebral affection: rabiform actions, epileptiform cramps, motions made in a circle, and a rapidly fatal termination. Muscular manifestations are much more rare. In a dog, Trasbot noticed complete inertia of the animal and extreme suffering when the members were touched or their rays bent.

Cysticercus cellulosæ has also been found in the sheep, cat, deer, monkey, bear, and rat.

MIESCHER'S TUBES: RAINEY'S CORPUSCLES.

NATURAL HISTORY. Our knowledge is yet quite incomplete on the nature of the utricles of the muscular tissue which were discovered by Miescher in 1843, described by Hessling in 1854, and later by Rainey, in 1857. Meischer's tubes are fusiform, cylindrical, or filiform elements, which are developed inside of the muscular fibres; sometimes they have microscopic dimensions, at other times they appear under the form of small, whitish, very fine rods

(pig); in large animals they reach a length of one to two centimetres; generally they are disseminated, notwithstanding that they exist in considerable quantity; sometimes they are found gathered in masses of the size of a pea or hazel-nut. They are limited externally by a thick cuticle which, several authors have stated, possesses a ciliated coating; according to Leuckart, this so-called ciliated coating may be only the crushed cuticle, divided into a large number of small fragments. They contain numerous corpuscles which are reniform, agminated, and bathed in a homogeneous substance disposed like the meshes of a network; they divide the utricles of large size and give them an areolated appearance. When perforated, a purulent or milky exudation occurs; by compressing their surrounding membrane, they may be emptied completely.

Formerly we considered these corpuscles as fungi, or as agglomerations of lymphoid cells, or even as evolution stages of the cysticercus. In recent times they have been regarded as related to the psorosperms (gregarines), and have been given the name of "*psorospermic utricles*." But their exclusive localization in the muscles seems to weaken this assimilation (the gregarines are ordinarily only found in the epithelial cells of the mucous membranes, and in the skin and liver). The accumulation of reniform corpuscles inside of the sarcolemma, the want of Miescher's tubes in carnivorous animals, etc., have, too, induced us to consider these elements as differing from psorosperms. So long as the obscurity prevailing as to their nature is not dissipated, they must be considered as separate organisms, probably belonging to the group of protozoa. Their origin is absolutely unknown. Undoubtedly ingested with the food, they traverse the digestive mucous membrane in order to become located in the muscular tissue, particularly the muscles in the neighborhood of the buccal or pharyngeal cavities.

Blanchard has established the following provisory classification:

Class: Sporozoa. Order: Sarcosporidia.

First family: *Miescheridæ*—parasites of the striated muscles, comprising two kinds: first, *Miescheria* (thin, homogeneous membrane); second, *Sarcocystis* (thick membrane which is traversed by very fine canaliculi).

Second family: *Balbianidæ*—parasites of the connective tissue which are provided with a homogeneous thin membranous envelope. This family contains only the species *Balbiania*.

Rieck (according to Railliet) classifies in the species *Balbiana* the psorospermic utricles of ruminants. They are generally found in the interstitial connective tissue of the œsophageal muscles of the sheep and goat (Morot found psorosperms in the œsophagus upon 30.2 per cent. of pigs which he examined, his statistics being founded upon an examination of 900 subjects).

The species *Sarcozystis* is common in our domestic animals, especially in the pig. Ripping found the tubes of Miescher in all pigs which he examined; Kühn found them in 98.5 per cent. of animals of that species, and Koch in 8 per cent. of pigs which were slaughtered in Vienna. After the pig, the horse and ox are the animals in which we observe most frequently Miescher's tubes.

Rieck, depending on his own experience, admits that, in certain circumstances, Miescher's tubes may produce acute myositis in the beginning, which passes gradually into a chronic state and leads to degeneration of the muscular fibres. This myositis would be the result of the passage of sarcosporidian spores into the interstitial connective tissue, through the muscular substance. With the penetration of spores into the muscular fibres and their encystment the second period would occur—chronic myositis; cellular elements would be transformed into fibrillar connective tissue.

The researches of Rieck help to sustain the opinion which has been given by Siedamgrotzky, Laulanié, Brouwier, according to which chronic interstitial myositis, which is frequently observed in the pig, ox, and horse, would be occasioned by sarcosporidia, which are found so frequently in the muscular tissue of these animals. This opinion has been combated by Schmidt-Mühlheim, Pütz, and Eberth.

AFFECTED ANIMALS. The tubes of Miescher are found exclusively in the muscles of herbivorous and omnivorous animals, but especially in the pig, ruminants (ox, sheep, deer) and horse; they have also been found in the buffalo, reindeer, goat, hare, rat, mouse, the chicken, and a few other birds; their existence in carnivorous animals is yet to be demonstrated. They locate in preference in the muscles of the buccal and pharyngeal walls, in the œsophagus, tongue, and the muscles of the larynx and back of the mouth; nevertheless other muscular groups may contain these in large numbers (neck and shoulders, mouth, abdominal walls, hind legs, diaphragm, heart, etc.). Sometimes the presence of Miescher's tubes in the muscular tissue starts an interstitial myositis, which

is especially indicated by cellular infiltration of the perimysium. In other cases they give to the meat of the adult ox the clear coloration of veal.

In old horses and in buffalo the tubes of Miescher seem to exist almost constantly. In the pig, Perroncito found them upon one-fourth of the subjects. Out of 100 sheep affected by cachexia which were examined by Moulé, 99 were affected by them.

Symptoms. The muscles of herbivorous animals very frequently contain Miescher's tubes without any morbid manifestations being produced by these parasites; but when their number becomes considerable they occasion inflammatory alterations in the muscular tissue. In a sheep and goat, Dammann and Von Niederhäusern observed dyspnoëic troubles consecutive to inflammation of the muscles of the larynx and pharynx. In a bull, Brouwier noted a difficulty in rising and in walking; in a pig, Virchow observed paralysis of the posterior extremities.

Anatomical alterations in the meat of animals which have been slaughtered for consumption are frequent. In six pigs, Hertwig found psorosperms in such numbers that the preparations showed on the surface as much utricular tissue as muscular.¹ The meat was flabby and watery, and the section showed a slight greenish coloration in a few hours.

For differential diagnosis, see Trichinosis.

The consumption of psorospermic meat has never occasioned any accident in man. In small numbers Miescher's tubes do not exert any influence upon the quality of the meat, but the observations of Hertwig show that they lessen its nutritive value when they are found in considerable quantity.

¹ Pütz has related, in the pig, a remarkable case of pseudo-hypertrophy of a large number of muscles, which was produced by Miescher's tubes (Virchow; Archiv, 1888).—N. D. T.

SECTION II.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE ENCEPHALON AND ITS ENVELOPES.

THE interpretation of numberless symptoms which are determined by lesions of the encephalon requires a knowledge of some physiological facts which are established by experimental science. By means of results of anatomico-physiological researches which have been made lately, we may nowadays make a topographical diagnosis of some affections of cerebral origin in our animals. In veterinary science, however, the pathology of the encephalon is yet little advanced, and it must always remain much more obscure than in human medicine. The principal facts which follow are the result of work by Fritsch, Hitzig, Ferrier, Munk, Goltz, and others.

1. The *cortical substance of the brain* offers several centres for our consideration. A. Frontal circumvolutions preside over certain psychological functions. Hyperemia, anemia, inflammation, abnormal compression, and degenerating alterations of this region produce psychologic troubles. B. In the anterior and posterior central circumvolutions and in the para-central lobule isolated circumscribed motor regions exist (psycho-motor centres) which control the voluntary movements of the muscles of the face, the tongue, and the legs. The various alterations existing in these centres (hemorrhage, softening, tumor, embolus, etc.) occasion sometimes spasms, at other times paralysis of corresponding muscular groups: of half of the face, tongue, and fore and hind legs; these limited paralyses, characteristic of diseases of psycho-motor centres, are designated *monoplegia*. C. Occipital circumvolutions seem to contain the visual centre and the temporal circumvolutions the acoustic centre.

2. The *channels of communication* of the motor centres with the muscles pass through the inner capsule, the cerebral peduncle, the

pons Varolii and the pyramids of the bulb, upon the surface of which the conductor cords are crossed. All the fibres of the psycho-motor centres being reunited there in a thin fasciculus, any affection of the isthmus (which involves the territory lying between the ventricles and the pyramids of the bulb) leads to a contraction or a unilateral paralysis of all the muscles innervated by the above-mentioned centres (face, tongue, fore and hind legs). Paralysis of a lateral half of the body (hemiplegia) is characterized by an affection of the motor isthmus, which is mostly localized in the neighborhood of the central ventricle. Decussation of the pyramids of the bulb gives an interpretation of the fact that hemiplegias, as well as monoplegias, are located upon the half of the body opposite to the cerebral lesion.

3. The *corpora quadrigemina* are the points of origin of the optic and oculo-motor nerves: their destruction causes blindness.

The CEREBELLUM plays an important part in the co-ordination of movements. The various lesions which concern it, especially those of the *cœnurus cerebrealis*, produce vertigo and irregularity of the gait. Affections of the cerebellar peduncle determine forced movements (like movements performed in the riding-school; rotation).

We must here observe, however, that the alterations found at the autopsy are not always those which ought to exist according to the physiological principles just mentioned.

Hyperemia and Cerebral Anemia.

Cerebral hyperemia takes two distinct forms: it is *active* or *passive*; this latter is produced by the several obstacles which obstruct returning circulation.

Etiology. 1. *Active cerebral congestion* is especially observed in young animals, more frequently in the horse, dog, and sheep than in other species. Some subjects seem predisposed to it. Its principal determining causes are: violent efforts, forced running, training (horse); mental excitements during the period of heat, or shipping per railroad (railroad disease), or after a change of stable; also over-activity of the heart in cases of hypertrophy of this organ; commotions, wounds of the brain and its coverings, the action of solar rays upon the head for a more or less prolonged time (insolation), the heat of summer, damp and hot stables, sudden cold, sudden changes of temperature, dental eruption (espe-

cially in the dog and horse; the influence of this cause has been much exaggerated); an augmentation of vascular pressure in the carotids, in consequence of the obliteration of some large arterial trunk (thrombosis of the arteries of the legs or of the pelvis—intermittent lameness); also compression of the distended vascular structures (arterial capillaries) by an abnormal gaseous tension in the stomach and intestine (acute meteorization of ruminants, gaseous colics of the horse); over-feeding after a prolonged abstinence or when the exercise is insufficient; foods which are difficult to digest (legumes, summer wheat, rye, clover). *Secondary cerebral congestion* may appear in the course of a number of morbid conditions (diseases of the brain, infectious diseases, and canine distemper; poisoning by narcotics and acrid narcotics). Old cerebral diseases (encephalitis, dropsy of the ventricles) predispose to cerebral congestion.

2. *Passive hyperemia* has as principal causes a compression acting upon the jugular veins by a very narrow collar, laryngeal collars used for wheezing horses, an extreme extension of the head upon the neck and shoulders (at the time of training the harnessed horse), voluminous jabots (especially in the dog); non-compensated valvular lesions, extended pulmonary alterations (emphysema, induration), compression of the lungs by liquid accumulated in the pleural sacs (pleurisy, hydrothorax).

A possibility of congestion of the brain, notwithstanding that this viscus is enclosed in unyielding walls, is explained by the existence of the cerebro-spinal fluid in the lymphatic perivascular spaces; at the same time that the bloodvessels become dilated, the ventricles and perivascular lymphatic sheaths become contracted and expel their contents, on one side into the central canal of the cord, on the other into the cervical trunk.

3. Causes of *arterial anemia* of the brain are: cardiac weakness, compression of the carotids by tumors, ligation of these arteries, thrombo-embolic obliterations of the cerebral vessels, considerable losses of blood and a lessening of its most important elements (anemia, abundant hemorrhages, chlorosis, leukemia), the too rapid ebb of the blood which is contained in the brain (puncture of an abundant pleuritic exudate or of the meteorized intestine), augmentation of the intra-cranial pressure when the encephalon is found compressed by a collection of liquid (immobility); also by tumors or hemorrhages. A reflex spasmodic contraction of the

cerebral vessels (after a "commotion," for instance) may undoubtedly also produce cerebral anemia. Finally, in a great number of cases this is but an epiphenomenon of a number of diseases accompanied by general anemia. Passive hyperemia is constantly followed by arterial anemia.

Pathological anatomy. 1. *Cerebral hyperemia* is characterized anatomically by fulness of the vessels of the brain, of the meninges, and by petechial hemorrhages. The brain seems to have increased in volume; if we make a narrow incision into the dura mater, the compressed cerebral substance escapes at once out of it. The gray substance has a brownish coloration; the white substance shows a dull gray or yellowish-gray tint; on section, we observe a large number of petechiæ which are sometimes disposed in ribbons; these disappear on wiping off the surface of the section, which does not happen in the case of apoplectic centres.

In the more marked degrees of cerebral hyperemia, any serum which has transuded from the perivascular lymphatic spaces into the cerebral tissue itself compresses the capillaries. This *cerebral œdema* is apparent to the naked eye by the anemia of the organ, by a peculiar moist aspect of the sections, by a serous oozing upon the surface of the latter, and by a lessening of the consistence of the cerebral substance.

After repeated congestions there usually persist suspicious circumscribed cloudy tumefactions of the cerebral covering, also vascular dilatations and thickening of the ependyma. Perhaps the arachnoidal granulations of Pacchioni are but neoformations which are produced by chronic cerebral hyperemia.

2. *Cerebral anemia* is marked by a state of vacuity of the bloodvessels of the brain and meninges; the gray substance is clearer, as if washed; on section we observe infrequently some bloody points. In anemic cases of long duration the cerebral substance has a firmer consistence than in the normal state.

Symptoms. *Acute cerebral hyperemia* is marked by phases of excitement separated by comatose intervals. During the periods of excitement there are sometimes cerebral symptoms; at other times troubles of mobility or of sensibility dominate the scene. The manifestations by which they are accompanied vary with the degree of intensity of the hyperemia, its localization, the animal species, and the individual.

In the larger number of cases the attack appears suddenly, in

others it is developed slowly; it is first marked by indications of restlessness and later by rabiform symptoms. Most horses lean forward, supporting the head or chest against objects in front of them (it is in this way that the wounds are produced which are observed upon orbits, lips, and chest); some horses rear, paw with their forefeet in the manger, or upon the chain used to tie them; some kick without any provocation; they will grind the teeth, bite objects which they can seize, neigh and shake the head, etc.; others *pull like the fox* (backward), they may break the halter and suffer serious falls backward; others, again, suffer from a muscular trembling and even epileptiform attacks. All are anxious, are very sensitive to the touch, to noise, and to light. The ox will leave its manger abruptly, move about and bellow, he may also butt without aim, or against the manger or on the front wall; if he falls, he is seized by epileptiform cramps and convulsions; the jaws are contracted, the mouth is foaming, the head, neck, and shoulders are bent or contracted convulsively, the eye rotates in the orbit. The dog, which is very restless and excited, howls, bites, snaps at imaginary objects, performs unusual movements, and seeks to escape; at times he is subject to vomiting, cramps, and convulsions. Beside these psychical symptoms, which are sensory and motor, we observe also, at the beginning of the attack, certain other phenomena: the conjunctiva is injected; at times the expression is dull, at other times it has its usual vivacity; the pupil is contracted; the ophthalmoscope shows an active, well-marked hyperemia of the optic papilla; the temperature of the brain is increased, the pulse is full, circulation and respiration are accelerated.

The comatose state, which appears rapidly, very often within a quarter of an hour, is indicated by entirely opposite symptoms. The animals are depressed, somnolent; the head is held low or leans supported by the manger, the face is without expression. The animal does not pay attention to anything that occurs, takes abnormal positions or executes automatic movements (forward or rotary movements); the gait is staggering; the appetite is lessened, capricious; micturition and defecation are delayed; the pulse is sometimes accelerated, at other times slackened.

The *course* of acute cerebral hyperemia is very inconstant. A complete cure is sometimes produced within a few minutes, but the attacks may be repeated for hours, even for several days. In other cases hyperemia leads to encephalitis or to fatal apoplexy (vascular

lacerations or cerebral œdema). It may also be followed by immobility or vertigo. The *prognosis* must therefore be guarded.

2. *Passive cerebral hyperemia* is expressed by manifestations of the second stage of active hyperemia—phenomena of depression. There are, however, cases where the comatose state is interrupted by periods of excitement. Very often the clinical aspect is that of cerebral anemia.

3. *Acute cerebral anemia* is generally indicated by vertigo and syncopal phenomena, by cardiac weakness, a small pulse, vomiting (in carnivorous and omnivorous animals), dilatation of the pupil, paleness of the optic papilla (ophthalmoscopic examination); sometimes the syncope ends in death (nervous apoplexy). In some cases the symptoms of cerebral anemia are identical with those of acute cerebral hyperemia; they consist of phases of excitement alternating with periods of coma; this is especially observed in *chronic anemia*.

Differential diagnosis. Cerebral hyperemia and encephalitis are marked by symptoms which are similar; sometimes it is impossible to differentiate them. As a general rule, we must conclude as to the existence of the first when the trouble is not alarming, the attacks of moderate intensity, and the hyperemia slight or entirely wanting.

Treatment. 1. If consulted when acute cerebral hyperemia commences, the first indication to be fulfilled is to practise bleeding, but this should be avoided if comatose phenomena already exist. We must then resort to refrigerants, which should be applied upon the skull (ice, cold douches or compresses), and also endeavor by means of purgatives to produce a derivation upon the intestinal mucous membrane. Drastics should only be used in the beginning of the disease, and solely for young animals which are plethoric or in good condition; if administered later, when the exciting symptoms have disappeared, they increase and prolong the comatose state by the intense derivation which they produce; we have gathered on this point some personal observations which are demonstrative. We generally have recourse, therefore, to laxatives: alkalies, nitrate of potash, and tartar emetic in small doses, aiding the action of these agents by means of cold clysters. Cutaneous derivation by irritating frictions is but rarely indicated on account of the excitability and anxiety of the patients. We should, on the contrary, avoid all external excitements, and place the animals in a dark, cool, and well-ventilated stable. Horses ought to be placed

in a box stall. Finally, we must give cooling food which is slightly laxative; in the horse green fodder is especially recommended; also roots and bran.

2. The treatment of cerebral anemia comprises means which are the reverse of the preceding. The syncope must be combated with stimulants: ether, alcohol, wine, ammonia, camphor, coffee, caffeine, hyoscyamine, atropine, and energetic cutaneous derivations, etc. In chronic cerebral anemia we must remove the causes of general anemia.

ADDENDA.

SUNSTROKE AND HEATSTROKE. Authors have often confounded these two affections, which are, however, essentially different.

1. *Sunstroke* is a cerebral trouble which is produced by solar rays falling directly upon the cranium. In mild cases a slight hyperemia is developed, which is marked by the usual symptoms. In a dog which had been exposed a whole day to the influence of solar rays, Benjamin observed rabiform phenomena, which disappeared in twenty-four hours after refrigerant applications upon the cranium. In more serious cases we have to deal with encephalitis or cerebral apoplexy. Siedamgrotzky saw a dog die suddenly which was tied and exposed to the sun during a burning July day; at the autopsy he found the surface of the brain much injected and covered with small hemorrhages; a considerable quantity of bloody serum was accumulated in the dura mater and arachnoid; sections made into the brain substance and cord revealed a large number of petechial hemorrhages.

2. *Heatstroke* is occasioned by over-heating of the whole body and by excessive exertion. It is generally observed during the hot season in horses subjected to exhaustive work, and in oxen and sheep which stray long distances. Several of the cases described under the name of *sunstroke* as occurring in the horse belong to this group. We are not yet decided upon the nature of heatstroke and the mechanism of its fatality. Some see in it especially an alteration of the blood (destruction of red blood-corpuscles, acid reaction, accumulation in the blood of carbonic and lactic acids, urea, etc.). Others consider it as the result of thermic complications; the temperature is very high, and a turbid tumefaction of the cerebral ganglionic cells has been recognized. (The internal temperature may reach, or even surpass, 43° C.)

The symptoms of heatstroke in the horse are: weakness, fatigue on the least exertion, an abundant sudation, staggering gait, dyspnoea, anxiety, palpitations of the heart, acceleration of the circulation, a weak and imperceptible pulse, dilatation and later contraction of the pupil, falls, convulsive movements of the extremities, and finally death. At the autopsy we find the blood thick and black; heart and lungs are congested. The treatment consists in the application of cold douches and the administration of stimulants. The patients must be placed in a cool and shady place.

Cerebral Hemorrhage: Apoplexy.

Etiology. This condition is also designated "blood-stroke," "heat apoplexy." Cerebral apoplexy consists in the rupture of a cerebral vessel, accompanied by a more or less abundant hemorrhage. The discharged blood compresses and disorganizes the cerebral substance.

This condition has numerous causes. In some cases it is related to cerebral hyperemia or to encephalitis, its causes then being those of the latter diseases: violent exertions, excitement, increase of the activity of the heart, blood-stasis, plethora, sunstroke, etc. In other cases it is the indirect consequence of vascular alterations, among which we must especially mention fatty degeneration and atheroma of cerebral arteries. These alterations play the rôle of predisposing causes. The determining causes which intervene are the various circumstances capable of increasing the blood pressure in the brain. Traumatic influences (cerebral commotion) specially produce hemorrhages in the meninges, mainly between the dura mater and the cranium (hematoma of the dura mater).

In the brain, as well as in the other organs, emboli are frequently complicated by hemorrhagic infarcts; in the dog we have seen a remarkable case in which the condition was related to pulmonary carcinoma. Finally, cerebral hemorrhages may be produced during the course of certain morbid infectious or toxic conditions (anthrax, petechial fever, poisonings, etc.).

Pathological anatomy. Cerebral hemorrhage is quite frequently seen in the sheep, the ox, and the dog; it is rare in the horse. The regions of the encephalon where it is usually found are the neighborhood of the lateral ventricles, the gray cortical substance, the corpora quadrigemina, and the optical layers. At times the hemorrhagic islands are very limited (capillary or petechial

hemorrhages), at other times they occupy an extended area (apoplectic centres).

Capillary hemorrhages are small, rounded, or elongated centres, the size of which varies from that of a millet-seed to a pea. Sections made from their surface are streaked like marble; coagula formed by the extravasated blood disappear only when we wipe or wash the surfaces of the sections. When the discharged blood is collected in the perivascular sheaths the capillary hemorrhages are designated under the name of *dissecting miliary aneurisms*.

In cases of laceration of the small arterioles the apoplectic centres attain the size of a pea, a hazel-nut, or an almond; when the blood escapes from a large vessel it may destroy an extensive zone of cerebral substance. New centres are formed by small bloody masses, which are coagulated, soft, or doughy; the cerebral tissue which surrounds these is softened. In old cases of apoplexy the blood-clot is contracted and of a clearer coloration; the neighboring tissue is the seat of a yellow pigmentation, a result of the diffusion of hemoglobin.

The discharged blood and the destroyed cerebral substance become gradually reabsorbed; certain centres are transformed into cavities with smooth walls and are full of liquid (*apoplectic cysts*); in others the solid elements—corpuscles, fibrin, nervous elements—undergo retrogressive alterations and disappear, a connective-tissue proliferation results, and an island of cicatricial tissue is formed which preserves indefinitely a yellowish or ochreous coloration due to hematoidin (*apoplectic cicatrix*).

Symptoms. Cerebral hemorrhage, as a rule, occurs suddenly, without any preceding phenomenon. Its principal symptoms are an uncertain, vacillating gait, vertigo, trembling, and forward, side-wise, or circling movements. The animals lose consciousness and fall, then they are attacked by convulsions, especially in the legs; in most cases the brain is greatly affected. We observe frequently an intense congestion of the mucous membranes of the head; vascular ruptures may be produced as a consequence, causing epistaxis and buccal hemorrhages. The pulse is very weak and imperceptible, the respiration is dyspnoëic; involuntary micturition and defecation are not rare. Failures of muscular power may occur from the outset or within a certain time, and their manifestations at times betray distinctly the seat of the encephalic lesion. Among the principal are: paralyzes of certain muscular groups (monoplegias),

unilateral paralyses of the lips, of the tongue, of the muscles of the jaw, ears, eyes, and legs; of the optic nerve and consecutive amanrosis; of an entire lateral half of the body (hemiplegias) and abolition of the sensitiveness in a more or less extended domain, sometimes in a lateral half of the body (hemianesthesia).

As we have said above, monoplegias indicate a lesion of the motor centres of the cortical substance of the brain, and hemiplegias a lesion of the isthmus on the side opposite to the paralysis. Cases, however, are quite frequent where it is impossible to conclude from the symptoms which were observed as to what was the alteration by which they were produced; we can only draw from the first a presumption which is often contradicted.

Cerebral apoplexy sometimes produces death within a few minutes or hours, sometimes only after a number of days or even several weeks. A cure is rare and generally incomplete; paralysis persists as a rule.

Diagnosis. The diagnosis of encephalic hemorrhage is based upon the sudden appearance of serious cerebral symptoms which are accompanied by paralytic troubles. In pulmonary apoplexy (active, intense hyperemia and pulmonary oedema) respiratory troubles dominate the scene. The apoplectic form of anthrax exhibits the same manifestations as cerebral apoplexy; it can only be differentiated by microscopic examination of the blood and by the enzootic character of the trouble.

Treatment. The treatment of cerebral hemorrhage is unfortunately without effect in the large number of cases. Bleeding and local refrigerant applications are only advisable during the apoplectic stage; when this is passed these measures are more harmful than beneficial; then, as in anemia of the brain, we may resort to stimulants (ether, alcohol, camphor) in order to combat the depression of the cerebral functions. When the symptoms of encephalic hyperemia are prolonged, derivation upon the intestine is advisable in order to prevent hemorrhage or diminish its intensity. Cases of paralysis must be treated by massage, electricity, and strychnine. Internally we may try iodide of potassium in order to influence absorption of the bloody extravasations.

MENINGITIS, AND ENCEPHALITIS.

GENERAL CONSIDERATIONS ON ENCEPHALITIS. The different forms presented by encephalitis in our domestic animals may be grouped according to the anatomical or clinical point of view from which they are considered. Pathological anatomists distinguish the following forms :

1. Inflammation of the dura mater (*pachymeningitis*).
2. Inflammation of the meninges (*leptomeningitis*).
3. Inflammation of the cerebral substance (*encephalitis*). This comprises abscess of the brain and some of the cases of cerebral softening.

Concurrent inflammation of the brain and of its envelopes has received also the name of *meningo-encephalitis*. *Leptomeningitis* may be of a serous, purulent, croupous, or tuberculous type; according to its seat we recognize *meningitis* of the *convexity* and *basilar meningitis*. This classification, notwithstanding its exactness, cannot, however, at the present time, serve as a base for a description of inflammation of the brain and its meninges in our domestic animals. In these the clinical distinction of meningitis and encephalitis is very difficult—impossible, so to speak—in some cases, on account of the analogy and similarity of the symptoms of these two affections. Besides, they coexist very frequently, or the one complicates the other.

Most of the cases described in veterinary medicine as *encephalitis* were certainly only cases of *leptomeningitis*. Autopsy alone enables us to fix accurately the localization of the process. The *intra-vitam* diagnosis of a well-marked form of leptomeningitis is still more difficult (with the exception of *basilar leptomeningitis* of the ox; see Tuberculosis). Nevertheless, it is established that common serous basilar leptomeningitis is the most frequent form of inflammation of the cerebral envelopes; the purulent or fibrinous form seems to be extremely rare. The diagnosis “*pachymeningitis*” is relatively easy when there is a wound of the cranium, or an affection of the ear, etc.; but it is impossible to determine distinctly the zone of alteration, to define the affected regions, and to tell how far it is advanced in the meninges and in the cerebral substance—localized inflammation of the cranium.

Encephalitis can only be recognized under exceptional circum-

stances—when, for example, being circumscribed, as is ordinarily the case, it is accompanied by *central symptoms*, or when present in the case of patients affected by lesions which may become complicated by metastatic encephalic abscesses. Let us also point out that the group of symptoms are not always due to the existence of abscesses (cerebral hemorrhage, tumor, embolus), and that metastatic suppurations may involve the meninges as well as the encephalon itself. For these several reasons we cannot accept at this time an anatomico-pathological classification.

The clinical division generally adopted up to the present establishes in encephalitis the two forms, *acute* and *subacute*. It has been said that the first is accompanied by symptoms of excitement, and its course is rapid; the second is marked by phenomena of depression, and it evolves slowly. But these are only differences in degree which do not present anything essential. Besides, meningitis or encephalitis may under certain circumstances develop the acute or subacute forms, and between these two types, which are more or less characteristic, we observe all the possible intermediaries. Moreover, several processes may originate in the brain, producing the clinical symptoms of acute encephalitis; such are hyperemia, pachymeningitis, leptomeningitis of serous, purulent, or tuberculous form; also encephalitis, emboli, and certain neoplasms. As for subacute encephalitis, its domain is still greater; we will return to this subject in the remarks upon encephalitis of the horse.

The special pathology of the brain, like so many other questions of veterinary nosology, is to-day in a transitory stage. Ancient empirical writings and practices are giving place to enlightened descriptions derived from physiology, pathology, and pathological anatomy. In the meantime it would be well to abandon the division into acute and subacute encephalitis.

1. Encephalitis of the Horse: Leptomeningitis: Pachymeningitis: Encephalitis.

GENERAL REMARKS UPON SUBACUTE ENCEPHALITIS OF THE HORSE. This disease, which has been recognized for a long time, is marked by symptoms of cerebral depression, from which the name *comatose encephalitis* has been given to it, in opposition to that of *rabiform encephalitis*, which is applied to the acute type. It has also been designated by the expressions, *encephalitis accom-*

paniced by somnolence (Ryschner), *hot disease of the head* (Autenrieth), *acute immobility with asthenic character* (Ekert), *acute cerebral dropsy* (Gerlach), *semi-acute encephalitis* (Hering), *semi-acute headache* (Warz), *passive encephalitis* (Spinola), etc. This multiplicity of expressions sufficiently indicates that *subacute* is more frequent than *acute* encephalitis. We have thought it best to precede its description by a few general considerations.

In the horse this trouble does not in fact represent a pathological entity. The descriptions which have been given of it and the post-mortem examinations do not leave any doubt on this point. A large number of practitioners still designate under the name of *subacute* meningo-encephalitis all inflammatory affections of the brain. The following morbid conditions have been confounded with it:

1. *Acute serous leptomeningitis*, which in the majority of cases is very probably a basilar leptomeningitis. It may take both forms, the acute and subacute, consequently it may be expressed by phenomena of excitement or coma. It represents the principal variety of encephalitis.

2. Some other inflammatory or apyretic cerebral diseases: *encephalitis*, *pachymeningitis*, *passive cerebral hyperemia*, *cerebral hemorrhages*, and brain tumors.

3. Certain *alimentary poisonings* (mycoses) in which cerebral poisons are developed. Among foods by which they may be caused we may mention lupin, clover (see Lupinosis and Trifoliosis), also leguminous and some narcotic or acrid-narcotic plants (*Equisetum*, *Lolium temulentum*, *Taxus baccata*). Most authors attribute it to the leguminous plants (clover, vetch, etc.), and describe the gastric symptoms as prodromes of the cerebral disease (colics, inappetence, icterus). These phenomena indicate sufficiently that true poisonings are here produced.¹

4. Various infectious diseases, the nature of which is undetermined. The simultaneous appearance of the disease in several animals; the fact that it has often been ascribed by authors to damp and unclean stables; the absence of all inflammatory altera-

¹ The disease described under the name of *abdominal vertigo* is but a hyperemia of the encephalon or a meningo-encephalitis occurring as a complication of intestinal troubles and due to particular causes of a toxic order. As has been remarked by Prof. Sanson, of the Société centrale de Médecine Vétérinaire, the word *vertigo* has, in scientific language, a well-determined and proper signification which does not at all correspond to that which has been given to it in veterinary surgery up to the present time. (See *Vertigo*.)—N. D. T.

tion in the brain and its envelopes; the existence of turbid tumefaction or fatty degeneration of the liver, decomposition of the blood, and ecchymoses—these are facts which tend to the recognition of a primary alteration of the blood, this being complicated later by cerebral symptoms which have given rise to the idea of an independent encephalic disease. Nothing is known concerning the nature of the noxious matters, or in what manner they find their way into the blood. Winkler admits as the principal cause of subacute encephalitis a poisoning of the organism by peptones; these are said to originate by a transformation of the albumin of the blood, produced by a ferment contained in the leguminosæ. In this theory, which is but an hypothesis and only applied to certain cases, the author, in order to explain the disease, is obliged to consider it as a disorganization of the blood.

We will only mention here inflammation of the brain and its envelopes.

Etiology of encephalitis. The causes of encephalitis are essentially the same as those of cerebral congestion: excessive exertions, intense cerebral excitement, high atmospheric temperature, traumatism, etc. The etiological influences which interfere mostly are:

1. A continuous high temperature (damp and badly-ventilated stables), the keeping of animals in stables at a high temperature, exceptionally hot spring or summer days, stormy weather.

2. Abrupt changes in the conditions of life and the mode of keeping of animals (after sales, transportation per wagon or boat, change from mountainous regions to flat, low countries, changes affecting new army horses; exhibitions). Prietsch has seen a horse attacked by meningitis three times within two years; the disease coincided each time with the animal's sale. In new army horses and those which have been bought within a short time, besides the preceding causes, others may act: over-stimulating food, the starting of work, colds, defective stables, etc.

3. Exertions of traction to which such animals are subjected as were kept in the stable for some time and which had been fed abundantly. Observation teaches that encephalitis and hemoglobinemia are often found in the same stables.

4. Over-feeding. For a long time this has been considered as a cause of encephalitis. Leguminous foods have been particularly mentioned (peas, beans, vetches, clover), and food rich in protein (certain grains, summer rye). Formerly it was asserted

that the frequency of the disease in any country was in direct ratio to the abundance of leguminous food harvested in it; in regions where these plants were not cultivated it would be unknown. This statement is incorrect, but we must admit that exciting and abundant food which predisposes to plethora may cause encephalitis.

5. Various alterations of the cranium. Wounds or even common contusions of the cranial vault, suppurating osteitis, a purulent infiltration which occasions a pachymeningitis which is often complicated by leptomeningitis and eucephalitis.

6. Parasites, neoformations, emboli, and thrombosis of the brain. Bruckmüller, Mégnin, Siedamgrotzky, etc., have found in the eucephalon œstrus larvæ which had produced general troubles or symptoms *dé foyer* (special centres). Albrecht and Von Heill have also found there larvæ of the armed strongylus, also cœnuri and cysticerci (see Sturdy or Gid). Emboli occur mostly in the course of strangles and pharyngitis; at this point are generally developed circumscribed cerebral abscesses. Lustig has observed an eucephalitis which was due to thrombosis of the cerebral arteries.

Secondary encephalitis is produced by various general diseases, but especially by infectious diseases. Strangles and pharyngitis may become complicated by purulent encephalitis, also by emboli; septicemia and pyemia are often accompanied by suppurating leptomeningitis (arthritis of colts). It has also been seen in the course of contagious pneumonia and of variola (Röll).

Horses which have not reached the adult age are predisposed to eucephalitis; it is usually observed in animals which are from two to six years old; it is rare during very young or very old age. A first attack favors a return of the disease; immobile or dummy horses, or the offspring of such parents, are particularly subject to it. Nothing precise is known about the influence of race, but common animals seem, nevertheless, to furnish the largest number of cases of this affection. Army horses are only exceptionally affected—a fact which is certainly due to regular feeding and to hygienic care. The influence of the seasons is established by statistics: cases of encephalitis are more numerous in spring and at the beginning of summer than at any other period of the year. In some countries the disease exists in an enzootic form; there are stables where it seems to be permanent.

Pathological anatomy. 1. Acute PACHYMENINGITIS is generally suppurative. The dura mater is hyperemic and overrun with hemorrhages, its internal surface is dull and covered with a fibrino-purulent exudate; at times the dura mater has a muscular appearance. Chronic pachymeningitis is characterized by connective-tissue fibrinous proliferations which unite the dura mater to the cranial wall, sometimes by bony neoformations (ossifying pachymeningitis).

2. Serous LEPTOMENINGITIS of recent origin is marked by hyperemia of the pia mater, tumefaction and redness of the pia mater and of the arachnoid. Between the envelopes a reddish-yellow liquid collects (external hydrocephalus) which, when it is abundant, depresses the encephalic convolutions. The cerebral substance itself is œdematous on its surface, at times it is softened and doughy; a serous liquid oozes from its depressions. The surface of sections made into its thickness is more moist than in a normal state; it is of a uniform tint or strewn with hemorrhages. A clear serous liquid accumulates in the ventricles (internal hydrocephalus), this exudate being at times turbid or bloody; its quantity is very variable (we find ordinarily from 30 to 40 grammes; in some cases but 3 or 4 cubic centimetres). Out of twenty-five cases of acute hydrocephalus observed by Schütz,¹ in twenty-two the lateral ventricles contained 10 to 40 grammes of liquid, in one 50 grammes, in another 60 grammes, and in the last 82 grammes. When the exudate is more abundant, the optic layers and the trigeminal bodies are flattened, the ventricles are dilated, and the surrounding cerebral substance is infiltrated; the choroid plexus and œdematous choroidal substance form gelatinous, yellowish-gray swellings.

Purulent leptomeningitis is usually located upon the convexity of the brain. There we find hyperemia, petechiæ, and a tumefaction of the pia mater. Between the arachnoid and dura mater we meet with a flaky yellowish-gray exudate, which is quite dense, of croupous nature, and mainly formed by pus corpuscles and fibrinous granulations. The cortical substance is sometimes congested, at other times anemic, œdematous, or infiltrated with pus (meningo-encephalitis). The ventricles are generally empty.

Chronic leptomeningitis is characterized by adhesion of the

¹ Schütz: communication.

arachnoid to the dura mater, by milky tumefaction, thickening, and intimate uniting of the pia mater to the cerebral substance, into the thickness of which it sends thick fibrinous tracts; the veins, which are dilated, have their walls thickened; there exists at times œdema of the pia mater, which is accompanied by a serous exudation and atrophy of the cerebral convolutions; in some cases the ventricles contain a small quantity of liquid; the vessels of the ependyma are much dilated; this membrane itself is more or less thickened.

In leptomeningitis the quantity of the serous exudate is extremely variable. According to Hering, the quantity of cerebro-spinal fluid varies considerably even in the healthy horse,¹ but the differences are much more marked in sick animals. In four horses which had died from cerebral diseases the total quantity of this liquid ranged between 120 and 270 grammes (the cerebral fluid alone varied from 40 to 120 grammes); in three other horses which had not shown any symptoms of encephalic trouble there were found as many as 300 to 420 grammes. Hering concludes from this fact that the pressure exerted upon the brain by the cerebro-spinal fluid cannot be the cause of the nervous symptoms which occur during the course of encephalitis.

No direct communication exists between the cerebral vesicles and the sub-arachnoidal spaces (Menault, Perosino, Hering, Gurlt, Franck, Müller). The liquid of the ventricles cannot run off in cases of decapitation. Hence it follows that the post-mortem examinations which are made of the ventricles give an exact idea of the conditions which exist during life. There is a tendency to consider as identical with the ventricular exudate the sub-arachnoidal liquid which runs off at the time of severing the head.

3. ENCEPHALITIS is ordinarily partial or circumscribed; it is more rarely *general* or *diffused*.

a. Circumscribed encephalitis, which is *non-suppurative*, is characterized by irregular foci, which are more or less rounded, and of the size of a pea to that of a pigeon's egg; in some cases the inflammation invades a whole lateral lobe and is propagated to the neighboring tissues. The altered areas are hyperemic or studded with interstitial hemorrhages; the cerebral substance is softened. According to Schütz, the glandular bodies and the

¹ Hering: Repertorium, 1871.

ganglionic cells are tumefied and in course of granulo-fatty degeneration; the axis-cylinder is irregular, the myelin¹ is infiltrated with granulations. The encephalitic centres undergo maceration, infiltration, and softening; they finally constitute a soft mass, which is formed of degenerated nervous and ganglionic cells, also of leucocytes and free fat cells; this stage is designated *simple encephalitis* or *cerebral inflammatory softening*. (This inflammatory softening is distinguished by a leucocytic infiltration due to local nutritive troubles.) The inflammatory process may stop here, but it very frequently becomes hemorrhagic, a condition favored by fatty degeneration and tearing of the vessels contained in the inflamed cerebral territory. This state is known as *hemorrhagic encephalitis* or *red inflammatory cerebral softening* (in opposition to the simple red softening of apoplexy). The coloring matter of the discharged blood becomes gradually decomposed and takes a yellowish coloration (*yellow inflammatory cerebral softening*). The exudate and detritus of the cerebral tissue may become more liquefied or be reabsorbed; there is then produced, according to the case, a *centre of gray gelatinous softening* (*gray inflammatory softening*), a cyst, a *connective-tissue sclerosis*, or a *cicatrix*.

b. Partial purulent encephalitis ends ordinarily in the formation of CEREBRAL ABSCESES. It is generally developed at the expense of consecutive hemorrhagic inflammatory centres, or of septic or pyemic emboli. These centres gradually become enlarged and undergo purulent liquefaction; sometimes a barrier of granulations is formed upon the periphery of abscesses, and transforms them into a closed sac; the pus, which is of a creamy consistence, has a yellowish or greenish tint and a bad aspect. Cerebral abscesses are most commonly found in the horse in cases of strangles; they are also quite frequent in the ox.

c. Diffuse encephalitis is extremely rare. At the autopsy of a horse which had shown the ordinary symptoms of encephalitis, Friedberger found but a yellowish coloration of the white substance of both frontal lobes. Microscopic examination showed an infiltration of the neuroglia by numerous leucocytes, also a distention of the perivascular spaces by the same elements, as well as great dilatation of the capillaries.

Symptoms of encephalitis in the horse. Manifestations of encephalitis in the horse vary extremely; sometimes the morbid

¹ White substance of Schwann.

picture is dominated by symptoms of excitement, at other times by indications of stupefaction, and cases are frequent where these associated phenomena form a most strange *ensemble*. The intensity and seat of inflammation, the temperament and intellectual development of the individuals also impress upon the symptomatology very marked differences. On account of these circumstances it is impossible to trace a typical clinical picture of encephalitis; we must restrict ourselves to a description of several symptoms in particular.

1. Serious cases are generally announced by symptoms of intense excitement; sometimes, however, this is entirely wanting or is only manifested slowly. Restlessness and agitation appear suddenly in the stable or during work; often we observe rabiform symptoms. The animals push against the wall, rear, pull in a fox-like manner, break the rope with which they are tied, and throw themselves down. These uncontrollable movements may occasion wounds, contusions of the head and chest, fractures of the orbital arches, the hips, and the cervical vertebra. It is difficult, sometimes even impossible, to direct the patients; they advance at an uncertain gait and push against all obstacles; once out of the stable they refuse to return to it. The eye is wild, the pupil is dilated. Ophthalmoscopic examination shows the optic papilla to be red and its vessels dilated. The general sensitiveness is extreme; the touch, the voice, and noises cause intensification of the attacks. Contractions are produced in certain muscular groups, in muscles of the face (convulsive contractions of lips, grinding of the teeth), of the neck and shoulders, extremities, and diaphragm (hiccough). Entire horses frequently have an erection.

2. *Depression* succeeds the excitement or appears at the onset; in some very serious cases, ending rapidly in death, it is entirely wanting. It is produced by somnolence and stupefaction; the head is carried low or supported upon the manger; the patients lean against neighboring objects; the eyelids are half closed, as during sleep; the pupil is dilated; the legs are sometimes gathered under the body, at other times much spread, or again crossed; at certain moments they are subject to spasmodic contractions; the patients keep these abnormal attitudes and also the "encephalitic habitus" for hours, sometimes for half a day. They may lose their equilibrium and drop; if compelled to move, they stumble and fall. The gait is uncertain and staggering, and the extremities are

dragged upon the ground ; we frequently observe riding-ring movements for whole hours.¹ When the animals lie down it is very hard to make them return to the standing position, and in rising they often remain upon their knees for some time.

The sensitiveness is often considerably dulled. The patients are indifferent to all that is passing ; we may step upon the head, introduce the finger into the ears, and even the stroke of a whip does not affect them ; they do not try to rid themselves of the flies by which they are tormented. We observe at times paralytic symptoms (amaurosis, deafness, dysphagia, ptosis, paraplegia, hemiplegia).

3. *Fever*, more or less intense, exists in the majority of cases ; the temperature may rise to 40°–41° C. ; chills appear as soon as suppuration is established in the meninges or encephalon. In a general way hyperthermia is proportionate to the intensity of the symptoms of excitement ; in the comatose state the thermic elevation is often wanting. During the period of excitement the pulse is accelerated and full, later it becomes slower and loses its fulness ; its frequency and character at times vary considerably in the course of a day ; it may fall below the normal (26 to 24 pulsations per minute) ; when nearing death it is always accelerated. The temperature is irregular ; the mucous membranes of the head are injected, especially at the outset ; the optic papilla is congested.

4. In serious cases *inappetence* leads at times to death by inanition. In the benign form the appetite is more or less diminished ; prehension and mastication are hindered ; often the patients refuse a good quality of food in order to consume coarse fodder, which they take in big mouthfuls. During the meal they will cease to masticate, keeping a handful of hay between the lips or a bolus of food between the cheeks and teeth. When the pharyngeal muscles are paralyzed the food may take a wrong direction, fall into the bronchial tubes, and produce a traumatic pneumonia ; the peristaltic movements are weak, defecation and micturition rare ; the rectum is much distended by fecal accumulation.

5. During the period of excitement the *breathing* is accelerated, later it is slackened ; in some cases the movements of respiration

¹ A quite frequent and remarkable phenomenon consists in the difficulty these animals experience in taking the first step. They resist, but having once started they continue to advance straight, without being guided, until they come in contact with some obstacle.—L. T.

are less frequent than in a normal condition (the number of respiratory movements may be reduced to six per minute); they are at times accompanied by a snoring noise on inspiration. Acceleration of breathing coexisting with a general comatose condition may awaken the idea of pneumonia produced by foreign bodies.

Course. The course of encephalitis is sometimes very rapid; death may occur in less than twenty-four hours from cerebral apoplexy. But, as a rule, its evolution is slow, and the form which it will take is only well defined after a lapse of time varying from several days to a few weeks (acute and subacute type). The symptoms which occur are far from being uniform; at times we observe periodical rabiform manifestations which are followed by somnolence, at other times paroxysms separated by intervals of unequal duration; finally, in other instances the comatose state predominates, and is alternated by periods of excitement. In mild cases cure takes place within from two to four weeks; in others, when the disease does not kill before the fifteenth day, it passes into a chronic state (chronic dropsy of the ventricles). Often we observe alternations of remission and aggravation. The course of encephalitis is greatly influenced by the weather: spells of great heat are fatal; cool nights and rainy weather are favorable.

Prognosis. This is generally serious. A complete cure is only obtained in the proportion of 20 to 25 per cent. (9 cures in 36 patients, according to Ströbel; 5 in 25, Kühne; 13 in 49, Adam). The passing to a state of chronic hydrocephalus (immobility) is to be feared almost as much as the death of the animal. Among the possible consequences of the disease we must also mention vertigo, general paresis, epilepsy, amaurosis, deafness, monoplegia, and hemiplegia. A first attack predisposes to recurrences. Though the serious form almost invariably terminates in death, we meet with some exceptions to this. Hering has several times seen a decided amelioration produced toward the fourth week, when the patients, which were always down, seemed to be lost. Sometimes the animals succumb to traumatic pneumonia, to septicemia, or to pyemia consecutive to gangrene from decubitus, to inanition, and to thirst.

Differential diagnosis. According as the phenomena of excitement or of depression dominate the symptomatic *ensemble*, encephalitis may be confounded with cerebral hyperemia and hydrophobia, or with immobility (see articles relating to these diseases).

Treatment. The patients must be placed in isolated, dark, cool, well-ventilated stables, and preferably in a box stall. If the latter is not available, they may be set free in a barn, a shed, or even in a corral. There is no danger in leaving them in the open air during the night when the weather is favorable. We must then begin a local and general treatment. We apply cold water, snow, or ice compresses upon the cranium, or any refrigerant compound (potassium nitrate, sodium chloride, sodium sulphate, vinegar, or water). We use at the same time cold rectal injections. These remedies are continued as long as the symptoms of cerebral hyperemia persist. Later, when stupefaction becomes more marked, cold-water douches answer best; they have a double action, being refrigerant and stimulating.

Bleeding, which was formerly practised in all cases of encephalitis, is only advisable when there are symptoms of intense cerebral hyperemia; under all other circumstances it must be rejected. As soon as exudation has taken place bleeding only increases the anemia of the brain which is produced by the pressure exerted upon the vessels of this organ by the encephalic exudate.

We must seek to produce a derivation through the intestinal canal. Drastics are favorable at the beginning of the trouble, but only when the cerebral hyperemia persists and when the constitution of the patient does not contraindicate their use; in all other cases they are hurtful, as the intestinal derivation increases anemia and cerebral depression. In a general manner, but more particularly in cases of slight constipation, anodyne purgatives, especially alkaline, and small doses of calomel, have a very salutary effect, even when some depression exists. When their administration by the mouth is not possible we may resort to subcutaneous injections of physostigmine (0.1 gramme of sulphate of eserine). It has been tried to obtain resorption of ventricular and arachnoidal fluids by the use of hydrochlorate of pilocarpine, administered in hypodermatic doses, for several days at a time, in doses of 0.2 to 0.5 gramme, or at one time in a dose of 1 to 1.2 gramme (Klemm). Pilocarpine has been of service in the treatment of acute and subacute hydrocephalitis; but experience has demonstrated that, far from being a panacea, it is, on the contrary, quite unreliable (Nagel, Siedamgrotzky),¹ and if used in large doses may be dangerous.

¹ Nagel, Siedamgrotzky (unpublished communications).

Cutaneous derivatives (croton oil, oil of mustard, cantharides ointment, if applied in frictions upon the back of the neck or on both sides of the neck and shoulders) are hurtful during the period of excitement; our personal observations lead us to add that their efficiency is as yet very doubtful during the period of coma; we may say the same of cauterization and setons.

The patients have to be subjected to a liquid diet; they should be given bran, slops, green fodder, or turnips.

The prophylaxis consists in suppressing or modifying the causal influences which have been pointed out in the paragraph upon etiology.

[There is no disease of the brain more likely to be benefited by venesection than acute encephalitis. Bleeding should be the rule, as it is always useful—omission of it the exception; this is especially true in full-fed, plethoric animals habitually receiving highly nutritious food, and is necessary even in cases of debility and anemia, where it is more likely to do good than harm; the bleeding should be full, free, and sufficient—it should seldom be repeated. Purgation is very important, and, after one free purging, moderate catharsis is indicated every few days sufficient to keep the bowels lax throughout the attack. Counter-irritation is of undoubted service, and should be applied upon the sides of the neck and shoulders. Immediate and rapid improvement is often seen as the result of this treatment. Should the extremities be cold, mustard baths are indicated, and should be applied to the legs and belly.—W. L. Z.]

II. Encephalitis of Other Domestic Animals.

In other animals the etiology, pathological anatomy, and treatment of this affection are the same as in the horse; its manifestations vary according to the different species, as follows:

1. In the ox the excitement is marked by bellowing, a laborious, noisy respiration, abrupt movements of the head, and butting of the horns—violent enough to fracture these organs; the eye is fixed, the mouth gaping and foaming; we notice trembling, epileptiform cramps, riding-ring movements; some animals walk straight ahead, overthrowing objects they meet in their way; others climb into the manger or butt against the wall. Later we observe stupefaction, uncertainty of gait, abnormal positions, diminution of sensitiveness, falls, a general paralysis, amaurosis (Degive), etc. The course is

very rapid as a rule. Death is its usual termination, and occurs within a time varying from a few hours to several days.¹

Encephalitis of the ox may be confounded with catarrhal malignant fever (contagious headache). In order to establish a diagnosis we must be aware of the existence, in this latter disease, of lesions of the eyes, nasal cavities, and sinus.

2. In the DOG the prodromes of encephalitis are anxiety, restlessness, hyperæsthesia, hiding in the litter of the stable, etc. The patients no longer recognize their masters; some animals howl constantly; others cry as soon as an attempt is made to touch them; some will bite when we try to lift them, but no aggressive tendency has ever been observed against man or animals. Rabiform phenomena are rare; certain dogs, however, which are kept confined, bite the bars of their cage, they are uncontrollably agitated, and scatter their litter; if free, they escape and run at random. The eye is haggard, the conjunctiva injected, the cranium hot, and the pupils dilated; we may observe vomiting and epileptiform spasms. Later, the animals are stupified and plunged in coma. The disease ordinarily ends in death in a relatively short time; sometimes, however, the comatose state is prolonged, and we notice the appearance, as complications, of amaurosis, deafness, etc.

Encephalitis is often confounded with hydrophobia; there are cases where a diagnosis is only established by the autopsy. In encephalitis aggressive manifestations, alteration of the voice, and the ingestion of indigestible substances are wanting; in hydrophobia injection of the conjunctiva is less marked and the head is not so hot. The *Linguatula tænioides* and intestinal worms may produce a form of nervous distemper having symptoms resembling those of encephalitis.

3. In the PIG we observe rabiform manifestations; the patient raises himself against the walls of the stable, utters piercing cries, and grinds his teeth; at times he is seized with convulsions or epileptiform contractions; in other instances he performs circular movements or drops and rolls like a barrel; sometimes he leans against the walls or the objects within his reach; the surface of

¹ Under the name of *enzootic meningitis*, Westerner has described a disease which exists in animals of the bovine species in the western sections of the United States, where it is known under the name of "mad itch." Mayer is said to have observed it in 1869 and 1871, in a distillery stable, in oxen which were being fattened. (Amer. Vet. Rev., vol. xii.)—N. D. T.

the skull and the base of the ears are very hot, the groin is foaming. Later, the head is kept low, and stupefaction and paralysis gradually develop. Pregnant pigs often miscarry (Kolb). The usual end is death; recovery is exceptional, and when it happens a cerebral condition similar to immobility may persist.

4. The SHEEP carries the head stiff and inclined to one side; he pushes against the wall, turns, staggers; is sometimes taken with convulsions. Confusion with sturdy or gid [cerebral cœnurus] is frequent.

5. In the GOAT we observe also stiffness of the head, neck, and shoulders, grinding of the teeth and epileptiform attacks (Langress). In the sheep and dog the differential diagnosis of simple encephalitis from cerebro-spinal meningitis is always difficult.

CHRONIC HYDROCEPHALUS: CHRONIC DROPSY OF THE VENTRICLES (IMMOBILITY).

Chronic ventricular dropsy is common in the horse, where it constitutes the principal cause of immobility; it is exceptional in the ox, dog, pig, and sheep. In the equine race it is most commonly found in adult individuals; it is rare before the fourth year. The gelding seems to be particularly predisposed to it, perhaps on account of the arrest of development of the brain which is produced by castration—a fact which has been scientifically demonstrated in these animals. The encephalon, having been arrested in its development, must present less resistance, as in the case of any incompletely-formed organ, to the various pathogenic influences. Authors are unanimous in recognizing that subjects of common breeds, which are of weak constitution and lymphatic temperament, with heavy head and narrow forehead, are predisposed to chronic ventricular dropsy.¹

Etiology. 1. *Symptomatic immobility* may be occasioned by

¹ In France there are regions where this disease is very common. According to Maucuer, it exists "as a true enzootic" in some localities of the Alps and in the valley of the Rhône, affecting in preference mares and young horses from six months to three years old, more rarely mules. There are certain farms where all young horses become immobile at the end of one to two years, on which they were compelled to give up the raising of these animals. In localities where the disease is common cases of encephalic disease in man are frequently observed. (See Bull. Soc. centr. Vét., 1888).—N. D. T.

encephalitis accompanied by acute or subacute hydrocephalitis. When encephalic phlegmasia passes into a chronic state it gradually creates immobility from the fourth week.

2. The causes of *idiopathic immobility*—a form in which development is slow and independent of the preceding etiological factor—are as yet little known. An hereditary transmission, however, has been demonstrated; for a long time past experience has established that animals which are the offspring of immobile parents are frequently affected, at a more or less advanced age, with chronic ventricular dropsy. Curdt describes heredity in a case of immobility which had been observed in a two-days-old colt. It is still uncertain whether dropsy is due to an affection of the vascular plexus, to repeated hyperemia, or to a permanent augmentation of the blood pressure in the brain. As occasional causes authors have for a long time mentioned passive or active hyperemia. It is known that the brain easily becomes congested at the time of great heats, under the influence of excessive exertions, from over-feeding with grain and leguminous plants, in plethoric conditions, during the course of liver diseases, certain gastric troubles, heart and lung diseases which are accompanied by troubles of the circulation, when the jugular veins are compressed by the harness, etc.

Pathological anatomy. The lesions of chronic ventricular dropsy are more or less marked. The volume of the brain is increased, its convolutions are flattened, and its furrows obliterated. The cortical substance is anemic and shrunken; when it is removed down to the ventricular wall a kind of hernia forms; if it is perforated, a limpid serum which has accumulated in the lateral ventricles, and sometimes also in the median ventricle, escapes through the perforation. The former are more or less dilated, at times they communicate freely among themselves through the septum, which is perforated by the liquid. Compressed for a long time by the exudate, the optic layers, the corpora quadrigemina, and cornu ammonis are flattened (cerebral atrophy). The ethmoidal lobules are oftentimes excavated and reduced to a very thin membranous sac. The lining membrane of the ventricles is thickened and invaded with small connective-tissue neoformations; the neighboring cerebral tissue is anemic, pale, very dense, and sclerosed. The vascular plexuses are thickened, tumefied, and infiltrated; their vessels are dilated and undulating.

The symptoms are sufficiently explained by the anatomical altera-

tions ; a permanent compression is exercised upon the encephalon and the cerebral substance is partially destroyed.

Symptoms. The primary manifestations of immobility consist of disturbances of consciousness, conception, volition, sensitiveness, and in unusual movements or acts performed by the animals. Secondly, we observe modifications of the circulation, digestion, and respiration when the general condition is normal and when the slightest febrile reaction does not exist. We not only find in this disease various degrees of intensity, but its different symptoms, which are unequally accentuated, may become associated and constitute an *ensemble*, the forms of which are as strange as they are dissimilar. The most important in the horse are the following :

1. *Troubles of consciousness*, that is to say, of the *feeling of existence*, of *being*, which is marked by the physiognomy and external bearing. The animals are drowsy ; the eye, half closed by the upper lid, is without expression, haggard, and stupid ; the head, which is kept in a dependent position, is rested upon the manger, the lower flank or one of the knees is sometimes pressed against the wall. The legs are in an abnormal position, oftentimes gathered under the body, crossed one in front of the other, or resting upon one another. We are able to place the animals at will in certain attitudes, which they will keep for a variable time. Dulness is still more prominent when working. Stupor is always very evident after exercise.

2. *Complications of conception or of relations between ideas, also troubles of sensation and those produced by external objects.* The animals, which are indifferent to everything that surrounds them, no longer notice impressions communicated to them—a condition which is first made evident by the action of the ears ; these organs make frequent and varied movements whose direction does not correspond to that of the sound-waves ; one of the ear trumpets is often carried forward and the other backward. Other peculiar phenomena are seen during prehension and mastication. Sometimes we merely observe at variable intervals short suspensions of mastication ; at other times the animals take their fodder in big mouthfuls which they masticate hurriedly, and then stop suddenly, keeping a handful of hay between their lips.¹ Generally they seek food which is scattered upon the ground,

¹ It is this phenomenon which is commonly expressed by saying that the immobile animal *smokes his pipe*.—N. D. T.

and do not touch that in the manger or rack, which fact depends, perhaps, upon variations in the pressure exerted by the ventricular liquid upon the cerebral substance; while drinking they plunge the whole mouth and nostrils into the liquid; in some instances they *masticate while drinking*.

3. *Disturbances of volition.* These are marked in various ways. The patients are lazy when working, and they have to be constantly urged; they no longer obey the voice which commands them; at times they resist their grooms and do not respond to the whip; they will also continually throw themselves sideways, refuse to move backward, and do it only after a great deal of trouble; they rear quite frequently; some have a tendency to move in a circle.¹ During these actions the feet are lifted very high, as if there was occasion to cross a stream of water or to step over some obstacle.

4. *Disturbances of general sensitiveness.* As a rule, sensitiveness is diminished or destroyed. Stroking upon the crown, introduction of the finger into the ear, a slap of the hand upon the forehead, and pinching of the skin of the flank do not produce any reaction.

5. *Secondary complications of the circulation, digestion, and respiration.* The pulse is ordinarily soft and weak; the peristaltic movements and defecation are delayed or suppressed; the respiration is deep and more or less slowed.

Lustig and Esberg have considered hyperemia of the optic papilla and of the retinal vessels of the neighborhood (papillary stasis) a constant symptom of immobility. An ophthalmoscopic examination allowed us once to observe this hyperemia; we have quite frequently noticed a well-defined anemia of the papilla, and in the majority of cases found it normal. Similar examinations have been made by Eversbusch and Berlin. Out of forty-three animals examined, Hayne has observed hyperemia of the optic papilla but twice.

Course. The course of immobility is essentially chronic. In numerous cases the general condition of the subjects remains stationary for years, sometimes even up to the time of death; in others

¹ A very characteristic symptom of this condition is the dragging of the fore members along the ground during the movement of backing, and which is not seen in animals which refuse to back because of a pain in the region of the loins.

we observe alternations of improvement or aggravation of the condition. The paroxysms occur under the influence of various circumstances (violent exertions, heating food, damp stable, high temperature, etc.). During the hot season a more or less marked aggravation is the rule. But when the external conditions are favorable (a cool and well-ventilated stable, suitable diet and exercise, or moderate work) the symptoms of immobility may disappear for a time, which improvement has favored the belief of recovery. With Gerlach, we are not prepared to admit such a favorable termination.

Chronic ventricular dropsy is often complicated by cerebral congestion or encephalitis, especially when it has been developed as the result of acute or inflammatory hydrocephalus. Then the manifestations of immobility are more or less obscured by symptoms which are proper to hyperemia or to inflammation of the brain. We see periods of excitement, and rabiform attacks which may be transitory (cerebral congestion) or permanent (encephalitis). The term "rabiform immobility," formerly given to that complication, does not merit preservation. It is useless to give a detailed description of this morbid condition, the symptoms of which are identical with those of congestion or inflammation of the encephalon. Let us merely observe that chronic hydrocephalus, which is not a fatal disease in itself, generally kills the patients when it is complicated by encephalitis.

The phenomena produced by chronic ventricular dropsy in subjects of other domestic species (ox, pig, dog, sheep) do not differ essentially from those of immobility in the horse. The affected animals are dull, insensible, and stupid; at times they throw themselves sideways abruptly; the gait may be uncertain—*groping*; digestive disturbances are frequent, but no elevation of temperature is observed. We say generally of these animals that they are *stupid*; in the pig the disease has received the name of *stupidity* (*Dämlichkeit*).

Treatment. The treatment of chronic ventricular dropsy does not offer any chance of success. The numerous measures which have been recommended and tried are of little efficacy. Hayne has advised emptying the ventricles by puncturing them through the ethmoidal lobules. Quite recently Klemm has suggested hydrochlorate of pilocarpine (1–1.2 grammes); but this agent is powerless in chronic hydrocephalus; the cerebral atrophy produced by this affection is an irreparable lesion. Rational pro-

phylactic measures may prevent the formation of abscesses and complications of cerebral hyperemia and encephalitis. The animals ought to be placed in cool, well-ventilated stables; they should be treated with moderation, avoiding heating food, giving them bran, green fodder and roots, and once in a while we should administer laxatives. During hot weather alkalies are advisable.

IMMOBILITY AS A CAUSE FOR SETTING ASIDE A CONTRACT OF SALE. Considered from this point of view immobility belongs to the domain of legal veterinary medicine. We shall make here only a brief examination of it.

1. *Definition.* Immobility in the horse is a chronic, apyretic, and incurable disease of the encephalon, characterized by troubles of consciousness, conception, volition, and sensitiveness, which differ considerably according to their degree of intensity and the existence or otherwise of complications.

2. *Causes.* Its principal cause is chronic ventricular dropsy. But other pathological processes of the brain may occasion the symptomatic features of immobility. These are: external chronic hydrocephalus, pachymeningitis, and chronic leptomeningitis, adhesions of the meninges between themselves or with the brain and the cranial wall, depression of the cranium, chronic encephalitis accompanied by softening or scleroses, cerebral neoplasms and those of the vascular plexus, cholesteatoma (these neoformations frequently do not determine any symptom), psammoma, melanoma, proliferating oedema, and brain parasites (hydatids).

3. *Diagnosis.* This is simple when the affection is clearly defined; it is difficult when the symptoms are imperfectly outlined, and the opinions which are formulated in this latter case are sometimes at variance. The animals must be first examined while at rest, later when working (harnessed or mounted). We must not pay too much attention to the various symptoms in particular, but rather to their *ensemble*. An unwillingness to work is an important sign; it is constant in immobile horses, but some animals can perform for quite a long time a certain amount of work. Horses affected with hydrocephalus are dangerous to work in public streets; they are, moreover, predisposed to encephalitis.

4. *Differential diagnosis.* We must distinguish the following conditions from immobility:

a. *Cerebral congestion* and *meningo-encephalitis*. These two diseases are particularly recognized by an injected condition of the

mucous membrane of the head, an elevation of the temperature of the cranium, and periods of agitation. Acute hydrocephalus has as principal symptoms: a rapid course, a pronounced febrile reaction, alternations of depression and excitement, anæsthesia or hyperæsthesia, diminution of appetite, and modifications of the pulse. The autopsy reveals acute inflammatory alterations.

b. General serious febrile diseases, especially infectious diseases.

c. Certain diseases of the head which accompany dental eruption and shedding. Redness of the gums, injection of the mucous membranes, and the young age of the animals enable us to recognize the dental evolution.

d. The forms of sexual hyper-function, designated by the expressions "ovarian tic," "uterine immobility" (these are periodical and correspond with the periods of activity of the genital organs).

e. Gastro-intestinal catarrhs and diseases of the liver. These morbid conditions produce symptoms to which have been given the names of "gastric tic" (Magenkoller) and "hepatic tic" (Leberkoller).

f. Stubbornness.

g. A phlegmatic temperament, fatigue, advanced age, deafness, blindness.

h. Inflammation of the sphenoidal sinus and of the ethmoidal labyrinth (Lustig).

5. *Delay for the beginning of proceedings in order to set aside a contract of sale:* Saxony, fifteen days; Bavaria, Würtemberg, and Baden, twenty-one days; Hesse, Prussia, twenty-eight days; Austria, thirty days.

URNSICKNESS, STURDY, OR GID.

Turnsickness (cœnurosis) is the most important of the parasitic diseases of the brain. Particularly frequent in the sheep, it is rare in the ox, and still more uncommon in the horse, goat, and antelope.

NATURAL HISTORY. Turnsickness is determined by *Cœnurus cerebralis*, a parasite which may be found exceptionally in the spinal cord. Küchenmeister has demonstrated experimentally (1853) that it represents the cystic stage of *Tenia cœnurus*, which commonly affects shepherd and butchers' dogs. The sheep is infested by ingesting proglottides or eggs deposited with the excrement of the dog upon the grass of pastures. During damp seasons the eggs

preserve their vitality for a long time; during such years we find a large number of sick animals. The dog is infested by eating the brains of sheep infested with *cœnuri*.

In the stomach of the sheep the eggs of the *tænia* are developed as soon as their shells are dissolved by the gastric juice; the hexagonal embryos, becoming free, perforate the gastric or intestinal walls, and then emigrate toward the brain and spinal cord. We do not yet know whether they are carried along by the blood flow or whether their progress is by way of the connective tissue. Müller thinks that they reach the brain by the vascular system, an opinion which is supported by the frequency of verminous emboli in the cerebral substance; the basilar substance of the brain is always free from inflammatory alterations, while the latter are very frequent upon the convexity of the hemispheres; the dura mater has never been found perforated. When the embryos are encysted in any region whatever of the brain or spinal cord, they lose their hooks and become transformed into vesicles. At the end of fourteen to nineteen days the latter are of the size of a milletseed or hempseed; after twenty-six to forty-two days they attain the size of a pea; toward the fiftieth day they are as large as a hazelnut; and toward the third month they arrive at their complete development.

Pathological anatomy. The anatomical alterations vary considerably with the various evolution phases of the vesicles.

1. The *period of emigration* into the brain corresponds clinically to the stage of cerebral excitement. The surface of the hemispheres is furrowed by dark-yellow, cylindrical sinuous tracks about one millimetre wide, and which are developed in the reticular tissue of the pia mater. According to Müller, its attachments are formed by a central hemorrhagic clot surrounded by pus corpuscles; the lesion consists thus in a *circumscribed, hemorrhagic, purulent leptomeningitis*. On corresponding points of the dura mater we find lesions of fibrino-purulent pachymeningitis. The sinuous tracts indicate the mode of entrance of the *cœnuri*, which are found at the bottom of the ducts in the shape of vesicles varying in size from a pin's head to that of a pea (about a dozen are found as a rule; Huzar! has counted thirty). Deep in the brain there exist similar sinuous tracts filled with a purulent, creamy liquid (suppurative encephalitis). The embryo may penetrate into the vascular plexus and occasion tumefaction, infiltration, an ependymal proliferation and internal suppurative hydrocephalus. We observe at times in the

brain necrotic centres, which are from one to two centimetres in diameter, and very probably produced by verminous emboli. Finally, the cerebral substance and the plexus may be the seat of more or less extensive hemorrhages—true apoplectic centres.

2. At the *period of complete development* of turnsickness the vesicles appear of a size varying from a pigeon's egg to a hen's egg; their form is rounded or ovoid; in the spinal cord they are depressed, are stretched, and become cylindrical; their contents have the clearness of pure water; their internal surface is covered with numerous scolices (as many as 500), the wall being sometimes raised upon their surfaces. In some cases we find but one large vesicle; in others several exist (two to six), which are of small dimensions; they push the cerebral substance back and raise the cranial wall, which is thin, paper-like, and in some instances perforated. The cerebral substance in the neighborhood of the vesicles is anemic and sclerosed (cerebral atrophy). The big vesicles may occupy an entire half of the cranial cavity (Reboul).

In the subcutaneous and interorganic connective tissue, also in the muscles, heart, liver, lungs, kidneys, mesentery, etc., we quite frequently observe the *cœnura* in a state of small particles, which are of the size of a pin's head or a pea, and undergoing granulo-fatty or calcified degeneration. In the subcutaneous connective tissue we may also find the *cœnura* completely developed.

Symptoms. I. TURNSICKNESS IN THE SHEEP. In this species turnsickness has received the most varied denominations (*whirling, vertigo, clumsiness*, etc.). It is generally observed in lambs; it is exceptional in animals more than two years old. During wet seasons it exists in an enzootic state in flocks and causes serious damage. It is known that the embryo may pass from the mother to the foetus. In the sheep the evolution of the disease comprises three periods:

1. The *period of cerebral excitement*, which corresponds to the emigration of the embryo. This happens in the latter part of the summer and the fall. Its duration is from eight to ten days.

2. The *latent state*, lasting from four to six months.

3. The period of true turnsickness, which begins in winter or spring; its duration is from four to six weeks.

The symptoms of the first period are generally observed in only one-fifth of the affected animals (Müller); they vary with the number of embryos, and frequently are not noticeable. The

patients drag themselves behind the herd; they are depressed, weak, awkward; the gait is staggering, the face is dulled and without expression. In serious cases the cerebral troubles are much more marked; the mucous membranes of the eye are red, the cranium is hot, the animals keep the head low; they are downcast and weak, and the majority have a "phlegmatic gait," but others are anxious and extremely nervous; they start away suddenly and run as if they were demented, or else trot in a circle; at certain times they are attacked with vertigo, stumble, fall, and are subject to spasms and convulsions. Sometimes the nervous phenomena are still more intense; in some of the cases (5 per cent., according to Müller) they cause death at the end of four to six days; in the largest number, however, they decrease in intensity after eight to ten days; some animals recover (about 2 per cent., according to Zürn), but in the majority the trouble is only relieved; it persists in a latent state.

The second period—that in which the disease is stationary—is not accompanied by any appreciable symptom; the animals appear cured; by watching them closely, however, we may recognize certain disturbances of the cerebral functions.

The *period of turnsickness*, properly speaking, begins with dullness and stupefaction. Mastication is frequently interrupted; the patients stop suddenly; at times they are weak and stumble continually; the eye is cold and haggard, and the pupil is dilated. There are often characteristic modifications of locomotion; among these complications we may mention: 1. *Riding-ring movements*: the animals turn to one or the other side while describing larger or smaller circles. 2. *Whirling*: the fore or hind quarters execute a movement in a circle, one of the fore or hind legs serving as a pivot, during which the head and neck are kept in a downward position. 3. A *rotation* upon the longitudinal axis of the body: they let themselves fall and roll like a barrel from one side to the other. 4. The *side fall*: taken by vertigo, they stagger and fall on their side. 5. The movement of *trotting*: they start trotting, going directly forward, keeping the head low and lifting their legs very high. 6. Lastly, a few subjects keep their head high or thrown back during the trot; they stumble frequently, and drop or fall backward. In Germany these subjects are given the name of "sailers." An important local symptom is the existence, on the surface of the cranial wall, of a depressible or fluctuating

region, distinctly circumscribed, compression of which causes pain and convulsions. Death occurs within four to six weeks; it is produced at times by cerebral paralysis, at other times by the advance of weakness and cachexia.

When the *cœnura* are developed in the spinal cord they gradually produce symptoms of sprain in the back (*kreuz drehe*): paresis of the hind quarters, staggering or cock gait, the hind legs carried under the body; pain and falling, produced by pressure upon the cord(?), and complete paralysis of one or both hind legs, which are dragged upon the ground (hydatid paraplegia). Death results from exhaustion and marasmus. When there exists at the same time a vesicle in the brain we observe obscuration of the intellect and the abnormal movements which have already been described.

II. TURNSICKNESS IN THE OX. In the bovine species we may observe turnsickness in adult subjects, but young bulls and heifers are most frequently affected. It begins with difficulty of prehension of food, which is often suddenly interrupted; the patients are anxious, and their movements are slow; the head is deviated or carried sideways and upward by constant convulsive contractions; the eye is cold and glossy. The base of the horns and the forehead are hot, the pupil dilated, the respiration and pulse accelerated. To these symptoms are added difficulty in locomotion; the animals push against the wall and perform very distinct riding-ring movements; sometimes they fall abruptly in the stable and may fracture their horns; it is very difficult—even impossible—to make them move backward. Percussion of the skull, practised upon the forehead by tapping with the metallic back of a pleximeter, furnishes some information; it produces, as a rule, a duller sound on the surface of the vesicle, the region around which is the seat of exaggerated sensitiveness. The course is more rapid than in the sheep; its termination is the same as in that species. Ordinarily the animals are slaughtered for consumption. Generally toward May or June killing becomes necessary. Between the appearance of the first symptoms and the time when they reach their maximum intensity four to five months may intervene.

III. TURNSICKNESS IN THE HORSE. This is extremely rare. At the beginning it is shown by manifestations of immobility or encephalitis. We observe movements of whirling or ring actions, backing, vertigo, blindness, etc. In most cases death occurs rapidly.

Differential diagnosis. Turnsickness may be confounded, especially at the outset, with several diseases, particularly with encephalitis, œstrus disease (the *Æstrus ovis* penetrates at times into the cerebral substance), epilepsy, vertigo, blindness, purulent catarrh of the maxillary sinus (Zürn), and, in the ox, with echinococci. The *intra-vitam* diagnosis of echinococci is not possible, but the presence of œstrus larvæ is sufficiently characterized by the catarrhal process in the nasal mucous membrane and the frontal sinus; as to encephalitis, it is never observed in an enzootic state. When turnsickness is recognized in a herd, the diagnosis of cases which occur successively does not offer any difficulty; we run no risk of making a mistake by considering as turnsickness any cerebral affection which appears in animals of the same herd. The affection, which is due to the presence of cœnura in the spinal canal, is easy to distinguish from lumbar prurigo (*Traberkrankheit*) by the pruritus and constant scratching to which animals are addicted when affected by the latter disease.

Prognosis. In any case the prognosis is very grave, but it is relatively less so during the second stage of turnsickness. If not treated, all the patients perish; treatment enables us to save about one-third. Sheep which are in good condition ought to be slaughtered as soon as possible. Opinions as to the value of surgical treatment are much divided. With Müller, Damman, and others, we believe that operation would not prevent numerous losses when the disease exists in an epizootic state.

Treatment. This consists in extraction of the vesicles which are developed in the brain. This operation, which has been practised for a long time, is especially advisable in cases of sporadic turnsickness. Various circumstances prevent favorable results in the majority of cases; often the vesicle is too deeply situated to be reached; in at least one-third of the patients several vesicles exist in the brain; the instrument used may cause hemorrhages or suppurative encephalitis; lastly, in some operated subjects the cerebral functions are not completely restored notwithstanding the mildness of the consecutive symptoms, because the anatomical lesions and accompanying complications are too pronounced. The two principal conditions essential to the success of the treatment are an accurate diagnosis of the location of the cœnuric ventricle and a suitable operative procedure.

1. GENERAL INDICATIONS FOR A TOPOGRAPHICAL DIAGNOSIS

OF CŒNURUS. When the cerebral territory where the cœnurus is developed cannot be recognized by palpation or percussion we should notice the manner in which the head is carried and the character of the abnormal movements. In cases where the head leans sideways the cerebral hemisphere, the position of which has become inferior, generally contains the cœnurus. When this symptom is wanting we may establish a diagnosis based upon the following facts in clinical and anatomical pathology :

a. In patients which perform *ring actions* the vesicle is situated superficially in one of the cerebral hemispheres—in the hemisphere which corresponds with the centre of the circle described by the animal in its movements.

b. In those which perform the *right whirl* the cœnurus is located sometimes upon the surface or in the depth of the right hemisphere, at other times upon the floor of the left ventricle ; the localization is reversed in cases of *left whirl*.

c. In *trotters* it occupies the frontal lobe of one of the hemispheres.

d. In subjects *falling sideways* it is developed in the central lobe or cerebellum upon one or the other side.

e. In so-called *sailers* it is located between the cerebrum and cerebellum.

f. In animals performing *rotating movements* upon the longitudinal axis of the body it is located on the base of the cerebellum.

2. Among the *surgical operations* the two principal are trephining and puncture with the trocar.

a. Trephining. In the sheep a trephine crown of about one centimetre in diameter is used ; in the ox and the horse the ordinary crown is applied. Operation in the median line must be avoided on account of the vascular channels which exist on the inner surface of the brain. When the vesicle cannot be recognized through the cranial wall, the two points to be selected are found : the first, 12 millimetres back of the centre of the horn in males, and 16 to 20 millimetres behind the horned rudiment, or the depression which replaces it, in the female, and at least 5 millimetres from the median line—a region which corresponds with the centre of the posterior cerebral lobe ; the second, immediately behind the internal limit, or of the sinus which takes its place—a region which corresponds to the superior portion of the anterior lobe (on the subject of trephining, see works on surgery). Zürn recommends

extracting the vesicle by means of a whalebone sound; this done, he advises searching if others exist, and, if any are found, to remove them in the same way. The edges of the cutaneous wound must not be joined by means of a suture, but by means of an em-plastic bandage, which is replaced by a dressing with turpentine bandage. When the bones of the skull are thin, the trephining instrument may be replaced by an ordinary bistoury.

b. Puncture with the trocar. In Zeden's operation we introduce the trocar on either side, a finger's width behind the horn; the stylet should be withdrawn to allow the liquid contents of the vesicle to run out; the latter may be completely emptied by means of an exhasting apparatus. When the canula has been withdrawn the wall of the vesicle is pumped simply by means of a syringe and extracted with a rat-toothed forceps. Damman has modified this process: he introduces the trocar to a depth of one centimetre, and does not penetrate further unless the vesicle has not been reached; in addition, he inclines the point of the instrument slightly inward. If necessary, we operate in the four regions which have been specified above (on the right and on the left, behind the horn and upon its internal angle); we may even puncture the centre of these four selected points, somewhat near the median line. In the *modus operandi* recommended by Erdt, after having made an opening in the cranial wall by means of a special perforator, we introduce the trocar obliquely, from front to back, a finger's width behind the base of the horn and at an equal distance from the median line; the slits of the canula are dentated with the object of hooking the cœnurus and extracting it more easily.

Prophylaxis is of far greater importance than the curative treatment. It consists in eradicating the causative agent as completely as possible. We administer anthelmintics to the shepherd dogs affected by tænia (this measure should be resorted to several times a year) and destroy the encephalon of sheep affected with turnsickness; such are the two principal preventive indications. In countries where this disease is of frequent occurrence a regimen of permanent stabling would be advisable for the lambs, notwithstanding the expense entailed. As a prophylactic indication, it has been advised to destroy foxes, wolves, and weasels, animals which are sometimes infected by tænia, but this measure is evidently of only secondary importance.

ROLLING DISEASE in the dog has a symptomatology analogous to turnsickness in the sheep.¹ It is characterized by attacks during which the patients suddenly move the head to one side and perform rotary movements of the whole body upon its longitudinal axis. Troubles of intellection seem to be wanting. These phenomena, which indicate a disease of the cerebellum, may be due to various and unknown causes. In some cases we have found at the autopsy, in the temporal lobe and cerebral peduncles, hemorrhagic centres of traumatic origin.

Rolling disease is also quite frequently observed during the course of the nervous form of distemper; it is then the expression of a lesion of the cerebellum or of one located in its neighborhood. In one case we found it as a symptomatic morbid condition produced by persistent constipation. According to Mauri, it may be caused by a cerebral embolus; in two cases this author has observed a circumscribed softening of the cerebellum.

Hypnone (0.25 to 2 grammes) and urethane (2 to 20 grammes) are the agents which have given the best results in the treatment of this disease.

CEREBRAL TUMORS.

Cerebral tumors, which are comparatively rare, are usually found upon the vascular plexuses, where they may develop unnoticed. We have recognized in these plexuses cholesteatoma, melanoma, psammoma, fibroma, and proliferating œdema. In the meninges have been found sarcoma and lipoma of the dura mater, epithelioma, and dermoid cyst. Finally, on the cranial wall, sometimes depressions are developed which, by compressing the encephalon, determine the same accidents as cerebral tumors. (Concerning tuberculous neoformations of the brain, see Tuberculosis; for parasitic tumors, see Turnsickness, Encephalitis, and Immobility.)

The manifestations of tumors of the brain are sometimes those of encephalitis, at other times those of immobility, at other times, again, those of apoplexy. Symptoms of *cerebral centres* alone, becoming gradually accentuated, when no internal or external cause exists to which they may be ascribed, betray these neoplasms, the topographic diagnosis of which is impossible. There are some which, being absolutely without symptoms, are not

¹ Mauri: Recueil Vét., 1871. Friedberger: Münch. Jahresber., 1877-78.

suspected during life. In the few cases reported in our literature there are mentioned as principal symptoms: a tendency to walk in a circle, phenomena of excitement, amaurosis, unilateral atrophy of the muscles of the jaw, hemiplegia, and circumscribed paralysis.

Participation of encephalic nerves in the disturbances is recognized as an important aid in establishing a diagnosis. Paralysis of the oculo-motor nerves, of the optic nerves, of the motor branches of the trigeminus (nerves of the masticatory muscles), and of the facial and hypoglossal nerves indicate a localization of the tumor at the base of the brain. In man a blood stasis in the optic papilla is a characteristic symptom of cerebral neoplasms; nothing is known as to the existence of this phenomenon in animals. (Hyperemia of the papilla is a consequence of the increase of cerebral pressure by the tumor; the cerebro-spinal fluid is thrown back into the lymphatic sheaths of the optic nerve; an œdema of the perforated lamella is produced, and, thus, its vessels being compressed, the papilla itself becomes the seat of a passive congestion.)

LIGHTNING-STROKE.

This accident is not of equal frequency in animals of different species. Lightning selects special victims among animals which are led to pasture (ox, horse, sheep). Its consequences vary according to the intensity of the electric fluid, and also according to whether its influence is direct or indirect as experienced by the animals.

Symptoms. Violent electric discharges produce instantaneous death. Slighter strokes and those which fall upon a neighboring object cause certain conditions of stupefaction and paralysis. Sometimes they occasion loss of consciousness, which persists for several hours; in other cases they produce paralysis of several members or of the hind quarters; the latter are completely paralyzed in some instances, in other cases are affected by paresis only; in the latter case the consequences of the accident are usually slight. A stroke of lightning quite frequently causes external wounds. On the skin we may find straight or angular lines or irregular figures, indicating the course followed by the electric spark and on the surface of which the hair is burned (these marks have been mistaken for imprints of twigs or branches). Lightning may also burn a large surface of the protecting hairy growths—

eyelashes, hair-tufts (Böllmann). We sometimes observe very deep burns of the hide and of the subcutaneous connective tissue and muscles; the latter, when lacerated, present a dark red or blackish tint.

Pathological anatomy. With the exception of local alterations, post-mortem examinations present nothing characteristic. In the majority of cases the cadaveric rigidity is slight; the venous system is engorged with dark liquid blood. The internal organs (brain, kidneys, lungs, etc.) contain numerous small hemorrhages; these are also found under the serous membranes. Sometimes, however, these alterations are entirely wanting. Decomposition of tissues takes place very rapidly.

Treatment. This is purely symptomatic. Stupefaction is combated with stimulants (camphor, alcohol, ether, ammonia, carbonate of ammonia, veratrine, atropine, caffeine, and hyoscyamine). Paralytic troubles require the use of muscular stimulants and irritating topical applications (frictions, massage, electricity, local irritants, and vesicants).

ADDENDUM.

Progressive Bulbar Paralysis.

Bulbar paralysis in man consists of atrophy of the motor nervous centres and of the medulla oblongata (the "bulb"), and especially of the centres for the hypoglossal, facial, and vagus. This atrophy of motor centres produces paralysis and atrophy of the tongue, lips, muscles of the jaw, face, velum of the palate, larynx, and pharynx; these various failures lead to interference with phonation, mastication, and deglutition, to ptyalism, and to a diminution of reflex excitability. We may also witness the occurrence of traumatic pneumonia as a consequence of dysphagia.

Degive, Gérard, and Laridon¹ have noticed a similar disease in the horses of western Flanders. These authors have observed atrophy and increasing paralysis of the muscles of the tongue, lips, and jaws, accompanied by difficulty in prehension, mastication, and deglutition (salivation, laborious deglutition or dysphagia, ejection of the alimentary bolus); the animals became emaciated, the masticatory muscles were atrophied, the reflex excitability was nearly *nil*.

¹ Degive, Gérard, et Laridon : Ann. de Bruxelles, 1883.

The disease developed very slowly; death occurred only at the end of six months or perhaps a year. As a complication, traumatic pneumonia was observed. All methods of treatment were unsuccessful. At the autopsy were found decoloration, atrophy, and fatty degeneration of the muscles of the tongue and lips, and a slight diminution in volume of the roots of the bulbar nerves; the medulla oblongata appeared normal. In man the ganglion cells of the motor centres are affected by degeneration, and the medulla oblongata is invaded by a connective-tissue neoformation.

II. DISEASES OF THE SPINAL CORD AND OF ITS ENVELOPES.

The pathology of the spinal cord and spinal meninges is still very imperfectly known in veterinary medicine. Observations on this nosological group are rare and much less precise than in human medicine, this fact is particularly due to the absence of any objective symptoms in the different diseases of the cord and the difficulty of their diagnosis in animals.

At the present time we can give only a very incomplete description of these diseases.

CEREBRO-SPINAL MENINGITIS: SPASM OF THE NECK.

Etiology. Spasm of the neck, or cerebro-spinal meningitis, is a mixed disease, to which is assigned a place between diseases of the encephalon and those of the spinal cord.

The term cerebro-spinal meningitis clearly indicates a localized inflammation developed upon the encephalic membranes and the anterior part of the spinal meninges. In its nature, symptoms, and anatomical alterations this disease resembles spasm of the neck in the human species; in animals it must also be of an infectious nature, for, in a majority of cases, it takes on an enzootic or epizootic character; but we are ignorant as to the nature of the specific causal factor. We have studied it here because it is frequently confounded with encephalitis. The causes to which it was formerly attributed, such as cold, damp and chilly weather, heated stables, clipping, over-feeding, are of but secondary importance.

We must also point out that a large number of clinical facts have been erroneously reported under the name of spasm of the neck. Hydrophobia (Kolb), tubercular basilar meningitis (Meyer), apoplexy, simple encephalitis, and certain poisonings have been confounded with that disease. We have been guided in describing it by observations obtained with special care and showing most analogy with the similar disease in man. Like the latter, cerebro-spinal meningitis of animals is expressed by an inconstant clinical picture, which is variable in animals of the same species.

It was first observed in America about the year 1850, where it had been studied by Large, of New York. The first observations made in Germany date from 1865. In 1876 it existed in an epizootic state in the different provinces of Egypt. In Cairo and its suburbs it caused the death of 5000 horses, 700 mules, and 200 donkeys (Apostolides).

Cerebro-spinal meningitis, which is most common in the horse and sheep, is also observed in the ox, goat, and dog. Young animals, especially lambs, wethers, and strong colts, are predisposed to it. In the spring it assumes quite frequently an enzootic character; at this season animals of different species, but especially horses and oxen, are affected in large numbers.

Pathological anatomy. In the beginning the alterations are those of serous cerebral and spinal leptomeningitis; in a more advanced stage the inflammation is evidently purulent. Between the dura mater and pia mater of the brain, medulla oblongata, and spinal cord, as well as in the cerebral ventricles, we find a serous, yellowish, and turbid liquid or fibrino-purulent exudate, which is milky or yellowish-gray, and is especially abundant at the base of the encephalon. In the sheep Roloff has seen purulent infiltration of the pia mater. In some cases the nerve roots are entirely surrounded by purulent exudate. The superficial layer of the brain and of the spinal cord is at times softened by the cedema; at other times it is infiltrated with pus. We find also islands of softening in the brain and cord; there are also quite frequently hemorrhagic surfaces or centres of various extent located in the meninges and encephalon. The vessels of the cerebral and medullary pia mater are hypermic.

General alterations have been described which betray the infectious nature of the process. The blood was found to be black, of bad aspect, and non-coagulable; the liver was softened and of a clay

color; the heart and other organs were in course of granular or fatty degeneration; we have also seen hemorrhages in different organs and catarrhal lesions of the intestine and of the rennet in lambs.

Symptoms. The clinical picture of cerebro-spinal meningitis is formed by an association of indications belonging to encephalitis and to myelitis. The dominating symptoms are stupefaction and drowsiness (over-excitement is rare), which coexist with spasmodic contractions of the muscles of the head, neck and shoulders, and extremities; the region of the neck is stiff and distended, the shoulders and head are jerked convulsively backward (opisthotonos); as a rule, there is quite an intense fever. When the medulla oblongata is principally affected we observe frequently paralysis of the muscles of the tongue and of the pharynx, and dysphagia or absolute impossibility of swallowing (acute bulbar paralysis). The complications observed vary according to the intensity and localization of the process (brain, medulla oblongata, spinal cord) and the animal species. The same clinical forms are observed in man in spasm of the neck.

I. In the HORSE the disease is sometimes announced by a violent chill, in other instances by drowsiness and apathy. In some cases the animals stagger, fall, and show symptoms of encephalitis, especially great depression; in other cases we observe at first dysphagia; the patients continually make futile efforts to swallow the saliva, which hangs from the mouth in long threads (Burger-Coburg);¹ again, in other and rarer instances the disease starts with opisthotonos from the onset. Soon we observe stiffness and abnormal sensitiveness of the neck; the animals can neither lift nor extend the head; the neck and shoulders and sometimes the whole spine are stiff and distended; at certain moments we observe attacks of opisthotonos and trismus; the temperature of the skull and neck is augmented, the muscles of this latter region are distended, hard, and prominent. In a few patients we observe hyperæsthesia and anxiety. To these symptoms are added convulsive contractions of the facial muscles (tetanic facies), of the shoulder, and of the extremities, and clonic contractions of the ocular muscles (nystagmus); these phenomena are followed by trembling, falling, and muscular exhaustion. Later, paresis or paralysis of the hind quarters sometimes

¹ Burger-Coburg: (Unpublished communication.)

complicates the picture; the walk is staggering and awkward, and later becomes impossible; we may also observe paralysis of the optic nerves (amaurosis), of the lips, ears, etc. The appetite is diminished or wanting; there is dysphagia and ptyalism. The urine contains albumin and a few red corpuscles; true hematuria may be found. In some cases the temperature rises to 41° C., in others the increase is slight or altogether wanting. The pulse may be accelerated or normal, or even lowered; as a rule, it is small and soft. The mucous membranes are red or yellowish.

The disease generally assumes an acute type; its average duration is from eight to fifteen days; more rarely it causes death in four to five days, sometimes within forty-eight hours. When it exists in an epizootic state its course seems to be particularly rapid in the animals which were first affected; we may then observe cases of "apoplectiform" death—the patients are suddenly overcome by convulsions (opisthotonos); they fall and die, showing all the symptoms of general paralysis. Returning attacks are frequent. The prognosis is very unfavorable; according to Hartenstine, the patients succumb in the proportion of 90 per cent.

II. In the SHEEP we observe a few prodromes: depression, weakness, apathy, abundant salivation, convulsive contractions of the lips, moving in a circle, etc. We soon find the animals stretched upon the ground and as if paralyzed; they are very sensitive to the touch, and are sometimes overcome by convulsive attacks: the head and shoulders are bent upward and backward; the head may be carried as far as the withers. We also observe spasms of the muscles of mastication, grinding of the teeth, shaking of the head, convulsions of the extremities, etc. The forehead and cranium are hot, the conjunctiva injected, and the pupil dilated. In lambs the thermometer often marks a lowering of the temperature (an unfavorable prognostic symptom).

Sometimes the disease is subacute and the patients die within a few hours or a few days; in some cases it persists from one to three weeks. In lambs and also in adult animals the usual termination is death. Out of 43 sick sheep which were observed by Schmidt, 41 died.¹

¹ Wischnikewitsch has described an enzootic cerebro-spinal meningitis which was observed in a flock of 1700 sheep on a farm in the neighborhood of Pultowa. It was very contagious and appeared in spells. As to its cause, the author ascribes the disease to "miasmatic agents." The principal symptoms which were observed were the fol-

III. In the DOG the initial stage is usually characterized by a deep stupefaction; delirium is much rarer. The animals are feverish and trembling; they stumble over any objects which may be lying upon the ground; they stagger and perform involuntary movements (walking in a circle); they also show symptoms of dysphagia. The cranial surface is hot; the muscles of the neck are prominent and hard; the neck and shoulders are thrown back convulsively; we observe generalized cramps which are interrupted by short remissions; these spasms produce rapid exhaustion and death. During the two or three days which precede the fatal issue the cornea becomes slightly affected. The average duration of the disease is nine days; its extreme limits are three and twenty days.

IV. In the OX cerebro-spinal meningitis is accompanied by difficulty of prehension and rumination; at times the patients are restless and nervous; they shake their heads constantly; at other times they are stupefied, plunged in coma; the muscles of the neck are distended, the head is carried high, and if we try to lower it the animals fall; at some moments we observe trismus, opisthotonos, convulsions of the members, lips, twitching of the eye; lastly, we recognize paralytic symptoms.

In the goat the indications are nearly similar.

Vogel and several other veterinarians have described, in the ox, an *epizootic spinal meningitis*, which is different from spasm of the neck. The following symptoms are those which are mentioned by authors: an abundant salivation, a difficulty of mastication, dysphagia, anxiety, stamping, paralysis of the muscles of the tongue and larynx, and rapid exhaustion. At the beginning the patients would lie down and then rise immediately, doing this continually, until they soon found it impossible to stand up. Death occurred from the third to the fifth day. These phenomena seem to be produced by a disease of the medulla oblongata (acute bulbar paralysis). We have never seen cerebral troubles, spasms, or convulsions.

lowing: weakness, apathy, anorexia, suspension of rumination, frequent micturition and defecation; later on the gait became staggering, the animals lay down not to rise again; they were affected by convulsions; the eye was haggard, the head bent and thrown upon the body. Death occurred from the tenth to the twelfth day. At the autopsy we observed intestinal catarrh, hyperemia, pulmonary splenization and hepatization, a discharge in the meninges, tumefaction of the pia mater and of the dura mater, adhesions between these two membranes, a liquid collection in the ventricles, circumscribed yellow softening of the brain and of the spinal marrow. The cerebral and medullary substance, the exudate of the meninges, and the blood contained bacilli (Veterinari Westnik de Charkow, 1889).—N. D. T.

Epizootic spinal meningitis of the ox does not constitute a well-defined nosological species; with it have been confounded certain poisonings of mycotic nature, and also several other diseases. The case reported by Zipperlen was merely one of mycotic enteritis; the autopsy, in fact, revealed an intense inflammation of the stomach and intestines. Mycotic enteritis (see Vol. I.), like meningo-encephalitis, is accompanied by salivation, restlessness, exhaustion, paralysis of the tongue and muscles of the larynx.

Differential diagnosis. 1. In the horse cerebro-spinal meningitis has often been confounded with ordinary encephalitis; the error is so much the easier from the fact that the latter is sometimes complicated by spasms and convulsions, but it may always be avoided when the symptoms of spinal disease are quite pronounced and dominate the picture. 2. In the ox it is difficult in some cases to differentiate it from tubercular meningitis; the distinction is based upon the enzootic character of the disease and the existence of symptoms produced by certain localizations of tuberculosis (pulmonary or mammary tuberculosis, emaciation, and cachexia). 3. In the dog it may be simulated by hydrophobia and distemper; but by taking into account the *ensemble* of observed symptoms we may succeed in making a differential diagnosis in the majority of cases. 4. In the sheep, if meningo-encephalitis and turnsickness have certain characters in common, which may leave the practitioner in doubt, the diagnosis is established by the autopsy of the first animals which die or are killed. 5. Tetanus is distinguished from spasm of the neck by the permanent character of the contraction and the existence of cerebral symptoms.

Treatment. This is the same as that of encephalitis. We must resort to applications of ice to the head and to cold donches, and endeavor to produce a derivation upon the intestines by means of laxatives. We may try antispasmodics (subcutaneous injections of morphine, bromide of potassium, chloral hydrate, and chloroform).

In a horse Johné obtained favorable effects by using hydrochlorate of pilocarpine (in doses of 0.6 gramme in hypodermatic injections). When the disease takes an enzootic character we must isolate the patients and disinfect the stables.

MYELITIS AND SPINAL MENINGITIS.¹

In the different domestic animals the special forms of meningitis and myelitis cannot be differentiated clinically. These diseases are determined by traumatic causes: blows, injuries upon the dorsal region, commotions or fractures of the spine, violent efforts of

¹ Under the title of *infectious paraplegia of the horse*, Com  ny has described a contagious disease, observed in 1887, in horses of cavalry regiments which were stabled at the military school in the Quartier Duplex. During the first few months it existed at the same time as typhoid fever, with which it was at first confounded. This disease is manifested under the form of paraplegia, which is more or less sudden or intense. It is distinctly differentiated from typhoid fever. Never, in fact, are the phenomena observed which characterize the latter: depression, stupor, hyperthermia. Its unique symptom is *paralysis of the hind quarters*.

It embraces the *subacute*, *acute*, and *foudroyant* ("thundering") types.

In the subacute form the patients retain their physiognomy and usual disposition; the appetite is preserved; but in the stable the hind quarters no longer perform any movements; in the horse the sheath is congested and the penis hanging; in the mare the lips of the vulva are slightly   dematous. If the patients are walked, the hind legs, which are weak and staggering, become crossed, the fetlocks become knuckled, and the toe is dragged upon the ground. No febrile reaction is noted; the rectal temperature is about 37   C.

The acute form originates, like the subacute, without any preceding phenomenon, and is sometimes first marked by lameness of one of the hind legs, at other times by paraplegia. This occurs almost from the onset. The walk is very laborious; the vacillation of the hind quarters and relaxation of the articulations are such that at every step there is a risk of falling. In the horse the penis is hanging; in the mare the vulva is somewhat opened and its lips are   dematous. There is no trace of fever; breathing is calm, the pulse full and regular, the beats of the heart are normal. The temperature hardly exceeds 37   C. Cerebral disorders are exceptional. In one case the animal showed vertiginous phenomena.

In the foudroyant form, which is almost exclusively seen in the mare, the animals, which may be overcome either in the stable or during work, stagger and fall, not being able to rise again. The hind quarters are entirely paralyzed; the physiognomy is not altered, the respiration is calm, the pulse full, and pain hardly indicated; muscular quivers are observed upon the buttocks, croup, flanks, shoulders, and knees. The general temperature is diminished. The lips of the vulva are tumefied and infiltrated; the stream of urine is much spread out (paralysis of the bladder). Paralysis extends more or less rapidly, restlessness and pain increase, the patients complain, the heart-beats become stronger, and the pulse weaker. According to the case, pain may be either quiescent or well marked. At times the temperature falls to 36   or 35   C.; at other times it rises to 40   or 41   C. In five to seven days the patients reach the condition of a skeleton. Death occurs from asphyxia. In acute and subacute cases amelioration becomes marked toward the sixth day, and recovery is complete within a lapse of time which varies from ten to forty days. In the foudroyant form of this affection, which is always fatal, the patients succumb rapidly (eighteen hours to six days).

The essential anatomical alterations are *congestion of the meninges, hyperemia of the medullary substance, and a softening of the lumbar bulb*. We find also a certain congestive condition of the encephalic meninges and cerebral substance, but these latter

traction. In a cow, Reindl has seen suppurating myelitis produced by a darning-needle which had been swallowed and had reached the spinal canal. Some authors consider cold and rheumatism as causes. Myelitis is sometimes the result of the propagation to the cord of an inflammation developed in its neighborhood (carious vertebrae). It may appear during the course of some infectious diseases (septicemia, pyemia, strangles, canine distemper).

Pathological anatomy. Its lesions are similar to those of encephalitis. In spinal leptomeningitis the pia mater and arachnoid are thick, tumefied, congested, and covered with ecchymoses; a serous or purulent exudate (hydrorrhachis) is collected between the dura mater and the cord; the surface of the latter is at times œdematous or infiltrated with pus. Like encephalitis, true myelitis is generally circumscribed in spots; it is characterized by infiltration and softening of the medullary substance (red softening), which is doughy, grayish-red, and strewn with hemorrhagic islands; abscesses are seldom found in it. In the chronic form the interstitial connective tissue becomes proliferated and the nervous elements atrophied (interstitial myelitis or sclerosis of the cord). Acute spinal pachymeningitis is generally suppurative. When it exists in a chronic state, cartilaginous or bony plates are developed in the dura mater (ossifying chronic pachymeningitis).

Symptoms. The complexity of the symptoms of myelitis agrees with those of the functions of the cord. This organ serves to give passage to the motor (anterior column) and sensitive (posterior column) nerve fibres to the trunk and extremities; it also contains the centres which control the functions of the bladder, rectum, and genital apparatus.

The principal manifestations of myelitis are :

1. *Troubles of mobility.* In some instances we notice phenomena of excitement, at other times paralysis. The gait is stiff and the vertebral column rigid; we observe clonic contractions; the animals stagger, fall, and are partially or entirely paralyzed (paresis or akinesis). Paraplegia occurs in the majority of cases; rising is

lesions are apparently a consequence of asphyxia; the clinical features indicate, at all events, that they are accessory.

Nothing is known about the causes of this serious disease. The researches made in order to establish its nature have been fruitless.

Revulsives, purgatives, and stimulants are the agents which have given the best results.—N. D. T.

impossible; the animals of the larger species remain recumbent—like inert masses; those of the smaller species drag their hind legs. Large doses of strychnine which are fatal to healthy subjects often do not occasion any convulsion in these patients. When the disease is located in the lumbar cord the paralytic troubles are limited to the posterior members and the tail; when the cervical cord is affected the four legs are paralyzed. In this latter case we always observe a contraction of the pupil.

2. *Disturbances of the sensibility—hyperæsthesia and anæsthesia.* This form is marked by intense sensitiveness to the slightest touch, by excitement and restlessness; the vertebral column especially is extremely sensitive. Small animals (dog) utter cries of pain; the horse and ox are affected by convulsions, and try to bite; we have seen a horse literally lifted off the ground by convulsions. Sometimes the slightest movements are accompanied by sufferings and complaints. Paralysis of sensitiveness is characterized by a loss of spinal excitability and of the reflexes: mechanical, thermic, and electrical irritations (needle-pricks, great heat, electrical currents of high intensity) do not produce any reaction.

3. *Functional troubles of the bladder and rectum.* At the beginning there are generally retention of urine and also constipation, due to paralysis of the muscles of the bladder and rectum. The accumulation of urine may produce vesical catarrh, pyelitis, and sometimes nephritis. Paralysis of the sphincters, which occurs later, leads to incontinence of urine and feces. In males we observe at first permanent erections and later impotence. We recognize also in the paralyzed regions a diminution of temperature, sometimes œdematous tumefaction, sudation, and muscular atrophy when the disease is prolonged for some time. Finally, permanent decubitus determines, upon the integument of prominent parts of the body, patches of dry gangrene.

Myelitis generally takes a chronic form. But in the majority of cases the animals are slaughtered without delay. Recovery is rare, and when it happens a paretic state of the hind quarters frequently persists.

Differential diagnosis. Myelitis must be distinguished from *rheumatismal hemoglobinemia*, a disease with which it has often been confounded. Many authors consider hemoglobinemia also as a disease of the spinal cord, and apply to spinal meningitis and myelitis the description of a disease which was formerly known as

"black strangury." *Paresis of the hind quarters* has often been taken for myelitis when observed during the course of muscular rheumatism, and also the paralytic phenomena which sometimes complicate cases of nephritis. It is not always easy to differentiate these affections. However, in most cases the diagnosis may be established. Nephritis is betrayed by alterations in the urine; in rheumatismal hemoglobinemia we have, as elements of diagnosis, hemoglobinuria, the history, and the rapid course; lastly, in rheumatismal diseases of the muscles there is never complete paralysis. Precise facts are frequently wanting in order to distinguish myelitis from medullary hemorrhages and spinal tumors; there are cases where a diagnosis is only established by the autopsy.

Treatment. At the beginning we must resort to refrigerants or to the Priessnitz compress, which is to be applied along the vertebral column. Later, when paralytic troubles predominate, vesicant frictions (camphorated alcohol, essence of turpentine, tincture of cantharides, croton oil, etc.) are advisable. In small animals electricity renders good service. Phenomena of excitement are combated by constant currents (galvanic current) and paralytic symptoms by interrupted currents (induced currents). In cases of paralysis in the dog and small animals we generally use Spamer's apparatus, applying the electrodes to various points (to both extremities or to the sides of the vertebral column), then alternately upon the extremity of the paralyzed member and toward the middle of the dorsal spine. The intensity of the current must be in proportion to the degree of paralysis; at all events, we must avoid producing great pain. At the beginning we may also produce a derivation on the intestine by means of laxatives. Internally, we use salts of strychnine. For the horse, the dose of nitrate of strychnine is 0.05 to 0.1 gramme; for the ox, 0.05 to 0.15 gramme; for the dog, $\frac{1}{10}$ milligramme per kilogramme of body-weight, or 0.001 to 0.003 gramme on an average (the hypodermatic method is much preferred to administration through the digestive tract). When the existence of a liquid collection in the spinal canal is suspected we may try pilocarpine. In order to avoid eschars of the skin, the larger animals should be placed in a sling without delay. Lastly, in the majority of cases, we must empty the bladder by compression or with the catheter, and clear the rectum of dung which has accumulated in it.

PARALYSIS THROUGH COMPRESSION OF THE SPINAL CORD.

Etiology. If compression of the spinal cord is determined in some cases by alterations of an inflammatory nature, there are others where it is produced by hemorrhages or tumors. Among these latter, which are very rare in our animals, we have especially found sarcomata of the dura mater (melano-sarcoma and sarcoma with fusiform cells), lipomata, and cœnures. Enostoses of the vertebral column and ossifying pachymeningitis occasion the same complications as these neoplasms. As to hemorrhages, they are usually produced by traumatism (fractures of the vertebræ, commotions, wounds made by firearms), by an alteration of the vessels, or by emboli.

Symptoms. Paralyzes which are consecutive to *hemorrhages* of the spinal meninges or of the spinal cord appear suddenly (spinal apoplexy); this character distinguishes them from those due to tumors or myelitis. Generally their traumatic origin is also revealed by the history and by a minute examination of the patients. The extravasated blood may become absorbed and a cure may occur, which never results in cases of medullar compression by a neoplasm. Paralytic troubles are quite frequently preceded by phenomena of excitement (convulsions). In an ox Kammerer observed a spasmodic attack of the neck which was associated with a hemorrhage of the cord.

Paralyzes which are caused by the existence of *tumors* compressing the spinal cord have a slow evolution; often their initial stage is marked by symptoms of excitement; they become gradually accentuated and complete within a varying time. Their course is fatally progressive; recovery never takes place. In a large number of cases the symptoms are similar to those of myelitis.

Differential diagnosis. The following facts enable us to distinguish paralyzes due to a lesion of the cord from those produced by a cerebral alteration:

1. Paralyzes of medullary origin are almost always paraplegias, and paralyzes of cerebral origin hemiplegias; this difference is explained by the small size of the cord as compared with the brain; the pathological process which affects the first usually involves its whole thickness. For the same reason, paralysis is generally

sensori-motor (posterior and anterior columns); in cases of serious medullary alterations all the muscles which are situated behind the lesion are paralyzed.

2. In spinal paralysis there are seldom any cerebral symptoms, and the functions of the encephalic nerves are not at all disturbed; the clinical character of cerebral paralyses is quite different.

3. Simultaneous paralyses of the bladder and rectum indicate a medullary localization of the causal alteration.

4. Medullary paralyses generally progress postero-anteriorly—they are *ascending*.

5. Trophic troubles in the affected regions (muscular atrophy) characterized by medullar paralyses: the ganglion cells of the anterior motor column of the cord exert a trophic influence on the muscles innervated by them, a property which does not exist in the cerebral motor centres.

In the dog akineses from medullary compression are quite frequently confounded with paresis or spinal paralysis due to cardiopathies (valvular lesions). In cardiac dilatations which are of recent date and consecutive to valvular alterations we frequently observe paraplegias which are produced either by venous hyperemia or arterial anemia of the cord, or by a thrombosis of the crural arteries developed during the course of the cardiac disease. Consequently in case of medullary paralysis in the dog it is always advisable to make an examination of the heart.

Treatment. The treatment of paralysis from medullary compression is the same as that of akinesis produced by myelitis (see the preceding article).

The term "*ataxia*" serves to designate inco-ordination of movements. In ataxia the various muscles do not function at the proper time; some act too early, others too late; at times some muscular groups only, in some instances the flexors, at other periods the extensors, enter into activity. There is no paralysis. One of the ataxias which is most frequent is *dry spavin* of the horse, an affection which in the majority of cases seems to be of medullary origin. Barrier and Weber have described ataxic movements in a horse affected by interstitial myelitis (atrophy of the nervous elements by connective-tissue hyperplasia).

LUMBAR PRURIGO OF THE SHEEP.

Lumbar prurigo of the sheep is a chronic, apyretic, congenital disease, the nature of which is as yet imperfectly known. It is marked by hyperæsthesia, weakness, paresis of the hind quarters, and by a progressive emaciation leading to cachexia. Its ordinary termination is death. The comparison between this disease and *tabes dorsalis* (gray degeneration of the posterior portion of the cord) in man is purely hypothetical, for up to the present time no constant anatomical alteration has been found in the spinal cord of subjects affected with lumbar prurigo.

Lumbar prurigo was unknown before the importation of merinos (1765). It is especially observed in flocks of improved breeds, and which contain precocious animals; it is very rare in native sheep or the blacks. Rams of the age of two or three years are predisposed to it; ewes and wethers are much less often affected. In Prussia and Saxony, where numerous flocks of improved breeds are found, the disease causes much more damage than in the extreme north of Germany. It is one of the most dangerous diseases of the ovine species.

Etiology. Among its causes we must mention: first, an hereditary predisposition. The disease is generally introduced into flocks by breeding rams which are already diseased or carry the germ of the trouble. Certain conditions of breeding, such as consanguinity, precocity, a delicate constitution of the animals, persistent in-breeding, the use of ewes which are too old for the purpose of reproduction, a sexual over-excitement or exhaustion of males used when too young for breeding; these are so many etiological factors which have been considered by authors. Haubner attributes the disease also to climatic influences: swampy soil, which is exposed to freshets, and where an abundance of aquatic plants grow; also sandy, barren soil; but the influence of these causes is at least doubtful.

Symptoms. In its beginning, lumbar prurigo is not expressed by any symptom which permits us to recognize it. The animals are anxious, very excitable; they tremble when any attempt is made to seize them; the movements of their members are abrupt, their flexion jerky: the eye is haggard, immobile; we observe convulsive movements of the head; at certain times this organ is thrown backward upon the spine; the ears go through unusual movements (a phenomenon which is especially pronounced during

the hot season). The duration of this first period is from four to eight weeks during winter, from eight to fifteen days during summer.

The second period has a duration of two to four months in summer, from two to four weeks in winter. It is marked by a general muscular weakness and increasing paresis of the hind quarters. The gait is uncertain, hesitating (hence the names "trotters," "trotters' disease"); the hind legs are kept apart and carried much ahead of the normal supporting line; at times we observe movements which are similar to those of string-halt; the patients can no longer gallop. To these ataxic symptoms are added those of lumbar pruritus; the animals rub themselves, bite the base of the tail, the croup, and back, sometimes also the hind legs and even the front legs; hairless spots where the integument is inflamed appear on the surface of the body; the wool is dry and tufty, a great many fibres being broken. Within a variable time we observe a gradual appearance of paralysis of the hind quarters; the patients fall frequently and rise only with trouble. Emaciation and weakness become gradually accentuated, and death occurs by exhaustion.

Differential diagnosis. Lumbar prurigo may be confounded with paraplegia produced by *cœuuri* and other spinal paralyses. Anxiety, pruritus, a frequency of the disease in males and improved breeds permit us to establish a diagnosis.

Treatment. The different means recommended in order to combat lumbar prurigo are of no efficacy. The prophylaxis comprises the following measures: exclude the affected animals from breeding, use rams which are at least two years old and in sufficient number to preserve them from sexual over-excitement and exhaustion, give lambs and wethers good food; finally, avoid prolonged in-breeding.

III. NERVOUS DISEASES.

PARALYSIS OF CERTAIN PERIPHERIC NERVES.

Etiology. Paralysis of the peripheric nerves, which is quite frequent in our animals, is generally produced by causes of a traumatic order: for instance, contusion of the *facial* in the neighborhood of the temporo-maxillary articulation at the point where it turns the posterior border of the lower maxillary; wounding of

the *subscapular*, which is produced by injuries or during work, especially when the horses are harnessed together, also from decubitus, etc.

[Laceration of the subscapular muscle is a frequent cause of paralysis of this character, and not infrequently it is of quite a severe form. Lesions also of the great psoas frequently result in a very unique form of paralysis of the crural triceps, due to complication of the anterior crural. Localized paralysis of the anterior extremity (partial) may also result from comparatively slight traumas over the course of the radial or musculo-cutaneous nerve. —W. L. Z.]

Affections of the neighborhood of the centre of origin, tumors (Lydtin), inflammatory diseases of the base of the brain, may also determine paralyzes. They are sometimes produced by cold (Trofimow), infectious diseases, disease of young age especially, and by certain poisons (paralysis of the facial, produced by digitalis).

I. PARALYSIS OF THE FACIAL is marked by inertia of the muscles of the face of the corresponding side; the upper lip and end of the nose are turned toward the healthy side, the lower lip is flabby, hanging, or deviated and shrivelled toward the opposite side of the lesion; food accumulates between the cheeks and teeth; the patient's eye is watery and remains constantly open; the corresponding nostril being paralyzed, violent efforts sometimes produce dyspnœa (Whitfield). The cause varies with the degree of paralysis. Recovery is its most common ending.

II. PARALYSIS OF THE TRIGEMINI (fifth pair) produces unilateral anæsthesia of the face, buccal cavity, tongue, and eye (on the corresponding side). The non-sensitiveness of the eye permits irritating foreign bodies to locate on its surface and produce inflammation and ulceration of the cornea. When the motor branch of the trigemini is affected the muscles of mastication are paralyzed; food, the prehension of which is difficult, accumulates in the mouth; the saliva hangs in threads, the lower jaw is sometimes hanging, the molar teeth are irregularly worn off, and the masticatory muscles become atrophied.

III. PARALYSIS OF THE SUPRASCAPULAR occasions a lameness of the shoulder (neuropathic lameness). It is characterized by atrophy of the abductors of the arm, of the supraspinatus and subspinitus. In walking, when the diseased legs have to support the weight, the shoulder is deviated outward and the elbow

carried away from the pectoral wall. This lameness resembles that which is produced by a rupture of the muscles of the shoulder ; but in this latter case these muscles react under the influence of electric currents, and we have also as a guide the inflammatory symptoms which complicate the laceration.

IV. PARALYSIS OF THE RADIAL, the nerve which animates the extensors of the leg, and especially the muscles of the olecranon, is marked by incapability of sustaining the animal's weight : the various bony rays are bent at the time of supporting the body. This flexion is not produced when we exert pressure with the hand in front of the knee. All paralyzed muscles are flabby.

V. PARALYSIS OF THE CRURAL reacts upon the femoral triceps. At the time of support the articulation of the stifle (patella) is abnormally flexed. When paralysis is of long-standing, the femoral muscles are atrophied.

VI. PARALYSIS OF THE GREAT SCIATIC leads to inertia of all the muscles of the posterior legs, with the exception of the triceps, which is innervated by the crural. The paralyzed muscles no longer react under the action of electric excitement, and in the end become atrophied.

VII. PARALYSIS OF THE TIBIAL is marked by an extraordinary flexion of the hock and fetlock at the time of use as a support. The paralyzed gemelli of the leg and flexors of the phalanges are flabby and incapable of producing extension of the cannon. The gait is undecided and the tibial region is relaxed. The movements of the member are accompanied by an exaggerated flexion of the cannon upon the leg (harping), and the foot is lifted exceptionally high. The metatarsal flexor is not paralyzed.

Treatment. Irritating applications, cold douches, electric currents, vesicant frictions, subcutaneous injections of strychnine, veratrine, etc. : such are the principal means for the treatment of these paralyses.

True *neuralgias* (violent pain in the domain of the affected nerve) have never been pointed out in subjects of our different species. This depends, perhaps, exclusively on the fact that the animals cannot communicate to us the sensations which they feel. In man neuralgias are frequent ; they are most commonly recognized upon the branches of the trigeminal, of the large occipital nerve (occipitalis major), of the brachial plexus, lumbar plexus,

intercostals, and large sciatic; neuralgia of this latter trunk bears the name of "sciatica."

In a horse, Friedberger¹ observed in the region of the neck and shoulders and the forearm an intense pruritus which could not be ascribed to a local cause, and which yielded to subcutaneous injections of morphine. The question whether this neurosis was a true neuralgia or a form of the disease known in man as pruritis has not been decided.

IV. NEUROSES WITHOUT ANY KNOWN ANATOMICAL ALTERATIONS.

TETANUS.

Etiology and pathology. Experimental researches which have been undertaken in later years (Carle and Rattone, Nicolaïer, Rosenbach, Flügge, Brieger) have established the infectious nature of tetanus. The specific cause of this disease is a bacillus which is very common, and appears in the shape of a fine, straight little stick, which is similar to a silk thread and provided with a small head (spore). According to Rosenbach and Flügge, it is found in the earth, in soil which is impregnated with excremental matter (stables), and in several other places.² It obtains entrance, as a rule, into the economy through a wound; under its influence organic albumin undergoes a series of transformations and generates ptomaines which possess properties similar to those of strychnine. Of these alkaloids Brieger has isolated tetanine, tetanotoxine, spasmodoxine, and another toxine which is imperfectly known.³

¹ Friedberger: Münch. Jahresber., 1877-78.

² The bacillus of Nicolaïer exists in very large numbers in the superficial layers of the ground. It has been found by Prietsch in hay dust, by Sormani in the excrements of animals which were fed on food soiled with earth coming from gardens, public roads, or manured fields; by Sanchez Toledo and Veillon in the dejections of animals which had eaten any unaltered food. It appears usually in the form of a long, weak bacillus, one extremity of which bears a colored swelling, and later a brilliant spore of a diameter which is two to four times larger than the body of the bacillus itself (Nicolaïer); but in the pus of tetanics it has sometimes the aspect of a thin, elongated, straight little stick, which possesses a slight mobility (Kitosato). It is an anaërobic bacillus, which is cultivated by itself or in an atmosphere of hydrogen, and grows rapidly at a temperature of 42° to 43° C. It may also be cultivated in a relatively single state and become accustomed to growing in a hardly rarefied air.—N. D. T.

³ Brieger and Fränkel have given to the tetanic poison the name of *toxalbumine*. Its composition appears to be that of albuminoid matters. Vaillard and Vincent

Bonome was the first to find the specific bacillus in tetanic animals. He observed it in a horse affected with tetanus consecutive to a contused wound that had been infected by dry mud, and also in a sheep affected by this disease after castration.

The transmissibility of tetanus to animals of different species and to man was demonstrated experimentally, in 1884, by Carle and Rattone. Attempts at inoculation made previously, especially in the horse, by transfusion of blood, had been unsuccessful. Carle and Rattone have transmitted tetanus to rabbits by inoculating pus from a tetanic human subject. A short time afterward Giordano communicated it to the guinea-pig and the mouse by injecting these animals with pus taken from wounds made by the castration of horses, and which were affected by tetanus. This author has also established that the blood of tetanic animals is not virulent, a fact which explains the constantly negative results given by transfusion. Beumer has recognized that inoculation is only successful upon fresh wounds; if practised on suppurating wounds entirely covered with proud flesh, it invariably remains sterile. The tetanic bacillus possesses an extreme resistance. The secretions and dry exudate of wounds in animals or human beings suffering from tetanus preserve their virulence for more than four months.¹

consider it as a diastasis which is closely comparable to toxine, which is secreted by a diphtheric bacillus, and the effects of which are limited to the neuro-muscular system.—N. D. T.

¹ The researches which were undertaken on tetanus have given, in these latter months, important results. Sanchez Toledo and Veillon have succeeded in obtaining in a pure state the bacillus of Nicolaïer. Cultivating it in several mediums (gelatine, blood-serum, calf and horse bouillon), protected from oxygen and at an intermediate temperature of $+14^{\circ}$ – 38° C., they have seen the development in cultures of small mobile bacilli, which are elongated in undulated filaments and show, from the tenth day, a sporulated form which is drumstick or bell-hammer shaped. It is at that evolutive stage that the tetanic bacillus possesses the most resistance to the various causes of destruction: it is neither killed by a temperature of 90° acting for a quarter of an hour, nor by a 5 per cent. *phenicated* solution acting for ten hours, nor by a 1 per cent. sublimate solution acting for three hours. These authors have also observed the passage of the bacilli into the general circulation. The bacilli remain massed in the tissues which surround the inoculating wound; the invasion of the blood is weak, slow, and inconstant. After death they continue to grow in the blood and viscera.

Vaillard and Vincent have recognized that pure cultures of tetanic bacilli or spores act only by the toxine which they contain. The microbe not only does not become multiplied in the tissues where it is deposited, but it disappears rapidly, and if, before inoculating it, it is deprived of the toxine with which it is associated, it does not produce any disease. It can only exert its noxious effects by acting in concert with certain chemical substances (lactic acid, trimethylamine, etc.) or with certain common microbes (*Microbacillus prodigiosus*, pyogenic microbes, etc.). If inoculated alone, it

The dog is refractory to tetanic inoculation (Rosenbach), when made either with secretion of wounds or with cultures. Clinical observation also teaches that tetanus is extremely rare in this animal.

The hypothesis of the equine origin of the tetanus of man, which is advocated by Verneuil, has been successfully combated by Roux. We do not understand how some German doctors can defend the theory of Verneuil. The following fact, which was related by Kotschau, is at least amusing: In a case of tetanus occurring in a woman after ovariectomy, this author, in order to explain the inoculation, goes so far as to ascribe it to the presence of a veterinarian who witnessed the operation !

ANIMALS AFFECTED. While tetanus affects all domestic species,

is rapidly encysted and destroyed by phagocytes; but if it is associated with other micro-organisms, like those produced in wounds under ordinary circumstances, the latter attract the phagocytes: they absorb all their activity and permit the tetanigenous elements to develop without interference.

Kitasato has found a means of permitting us to give animals immunity against tetanus. Rabbits which have been rendered immune may receive without risk an injection of 10 cubic centimetres of a virulent culture (5 cubic centimetres suffice in all cases to kill a non-vaccinated rabbit). They are also rendered non-sensitive to the action of tetanic toxines; they may be injected with a quantity twenty times greater than that which kills other animals without showing the appearance of any tetanic phenomenon. Kitasato has also observed that the blood of rabbits which have been rendered refractory to tetanus and the serum of this blood may destroy an enormous quantity of tetanigenous toxine. These same substances, if inoculated in mice, confer upon them a complete immunity against tetanus, and if injected into subjects of the species in which tetanus had been previously inoculated they prevent its development.

The works of Tizzoni and Cattani have given results partially confirming those obtained by Kitasato. These experimenters have recognized that freshly-prepared chlorine water, trichloride of iodine, and a 5 per cent. watery solution of carbolic acid destroy quite rapidly *in vitro* toxine of tetanic origin, but none of these substances has been sufficient to prevent or counteract tetanus. By a series of more and more virulent inoculations, and by beginning with minimum doses which were at times sufficient to determine slight tetanic manifestations, they have succeeded in conferring immunity upon animals which are little sensitive to tetanic virus (pigeon, dog). We may recognize the property which is possessed by the blood and serum of animals refractory to tetanic poisons, by the conferring of immunity to the dog (by the subcutaneous injection of a small quantity of blood-serum) as well as to the mouse (intraperitoneal injection of $\frac{1}{2}$ cubic centimetre of this same serum) as a condition; in order to obtain this result, inject this serum at least four hours before the inoculation of the tetanic virus. Rabbits and guinea-pigs did not acquire immunity by the injection of blood or of serum from a dog which had been rendered refractory; it could be transferred to the rabbit with the blood-serum of a pigeon which was vaccinated. These researches show that the serum of an animal which has been rendered refractory is not equally active in conferring immunity upon subjects of different animal species.—N. D. T.

it is particularly common in the horse, donkey, mule, and small ruminants (sheep and goat). It is very rarely met with in flesh-eaters. In warm countries, especially in tropical climates, cases of tetanus are much more frequent than in cold regions. At Saint Domingo it is feared to such an extent as to cause the price of geldings to be double that of entire horses (Wagenfeld). In a general way, however, it must be classed among the rare diseases. A report by Hering, referring to affections found in many horses of the Würtemberg army for a certain number of years, shows a single case of tetanus out of 3000 patients.

Strong and plethoric animals, and those belonging to distinguished breeds, seem to be predisposed to it. In the bovine, ovine, and caprine species, cows, calves, lambs, and bucks are those which are most frequently affected. It is rarely observed more than once in the same patient.

During certain years and at undetermined periods it may exist in an enzootic state in the sheep and the horse. These enzootics have been recognized for a long time, but formerly it was impossible to interpret them in a plausible way; nowadays they are explained perfectly well by their infectious nature; but the cause of the greater activity of tetanigenous elements at a given period is unknown.

FORMS OF TETANUS. We generally recognize in tetanus the *traumatic*, *rheumatismal*, and *toxic* forms. Notwithstanding that recent experimental researches tend to doubt the existence of rheumatismal tetanus, we have nevertheless thought best to preserve the ancient division.

1. *Traumatic tetanus* is far more frequent than the rheumatismal variety. It may complicate a great variety of wounds; clinical observation, however, shows that it has a well-defined predilection for some of these lesions. Those occurring most commonly are: contusions, lacerations, wounds made by firearms, wounds made with foreign bodies introduced into the tissues; nerve, tendon, and facial wounds; but particularly wounds of the extremities, traumatism of the hoof in the horse, crushing of the foot in the dog, and, in general, all narrow and limited wounds which are harmless in appearance. In the horse the street nail is especially to be feared, also pricking in shoeing, crown wounds made by iron calkins, operated corns, suppurating seams (toe cracks and quarter cracks), and lesions of the velvety tissue. This predisposition to tetanus, which is induced by

wounds of the hoof, depends upon the possibility of their infection by excrementitious matter. Spinola mentions also the danger of tetanus when there is an infected wound. In the horse wounds of the foot are undoubtedly its cause in a larger number of cases than is generally admitted. In the school of Dresden, out of nine horses dead from tetanus within a few years, Hartmann has constantly found either a wounding of the foot during the process of shoeing or a foot *injured by nails*. Cases of traumatic tetanus increase thus considerably at the expense of those which are related to the idiopathic form. Tetanus may originate when the inoculating wound has formed a cicatrix (*cicatricial tetanus*). Let us also mention as traumatic lesions which favor its development: wounds which are produced by saddle and harness, fractures of the vertebræ and cranium, burns, cutaneous fissures occurring during the course of *petechial* fever, and the penetration of foreign bodies into decayed teeth.

Tetanus is observed at times after certain operations, especially after castration. In the goat castration by a process necessitating an incision of the testicular envelopes is almost invariably complicated by a fatal tetanus; this is a fact which is well known to practitioners; therefore they generally castrate this animal by ligature or clamps, which are applied upon the intact covering, or by a subcutaneous ligature of the cord. We have seen tetanus exist in an enzootic state in animals which had been recently castrated, no matter what the operative process had been. It is much to be feared in young bulls castrated by the application of a cord upon the intact pouches or by a subcutaneous ligation of the cord. Formerly it was common in the horse after the operation for a hernia, also in straightening or amputating the tail; in the sheep it frequently accompanied clavelization and an application of the hellebore trochiscus. It has become rare since the application of antiseptic means, a fact which proves its infectious nature.

Tetanus is sometimes produced by the wounding of internal organs. In the cow it appears frequently after parturition or abortion; most of the cases observed in the bovine species are related to parturition (wounds of the uterus and vagina, laborious parturition, putrid decomposition of the placenta, etc.). In some cases it is consecutive to intestinal lesions (ulcers of the intestine—Hering). Here, evidently, the tetanic phenomena are the result of an infection.

In newborn animals, the colt and lamb especially, tetanus may have as a starting-point the umbilical wound when this is the seat of a septic infection. In lambs it is not rare to find it in an enzootic or epizootic state (*Tetanus neonatorum*: *T. agnorum*). We must ascribe to it a large number of cases which were formerly described under the name of *paresis* of lambs.

A form of tetanus has been described which is said to be produced by blows, falls, etc., without any apparent wound. Besides traumatisms, this has been ascribed to the action of cold (a sudden chill, a sudden change of temperature, exposure, etc.); but the influence of this cause is at least doubtful. In the Vienna clinic, Roll has rarely observed traumatic tetanus in isolated cases; almost always he has seen it in several animals about the same period. [Cold *per se* certainly cannot produce tetanus; it is possible that by diminishing the vitality of the tissues it may exert a certain predisposing influence by making a more fertile culture ground in the wound for the development of the micro-organisms.—W. L. Z.]

When lockjaw results as a complication of an external wound the time which elapses between its production and the appearance of tetanic phenomena is variable. The first symptoms occur within a certain number of days, exceptionally in twenty-four hours. Among the cases of traumatic lockjaw which have been published, there is but a single instance where the disease has originated six hours after the trauma; the longest incubation has lasted six weeks. After parturition the average duration of this trouble is from five to eight days; but here also the delay may vary between a few days and several weeks.

2. *Rheumatismal tetanus*, which is far more rare than the preceding, is said to become apparent after a sudden chilling of the cutaneous surface, especially after abundant *sudations* (draughts, cold rains, and being out in the open air during cold, damp nights, etc.). In most cases it appears two or three days after exposure to these causes; in others, after a longer time. Hamon has seen it develop eight days after a cold produced by a very heavy rain-storm. In young animals (lambs) cold seems to be a frequent cause of tetanus; stables which are located in high situations, and which are open and exposed to winds, are believed to be causative of this condition; also a driving out to pasture during rainy days, and cold after washing. We do not possess any positive knowledge of the pathology of rheumatismal tetanus.

[In the very nature of things such a condition as rheumatismal tetanus cannot exist. In endeavoring to establish such a complication we are forsaking the substance for the shadow. Cold cannot produce tetanus; it may be the cause of rheumatism, spinal meningitis, etc., which diseases in lambs and other young animals may be confounded with the former malady, but to assign to it such a baneful influence as the production of tetanus is certainly not the logical result of sound scientific reasoning or investigation. Tetanus is always traumatic; its agents cannot penetrate into the tissues except through a wound upon the cutaneous or mucous surface. Rheumatismal tetanus is a myth.—W. L. Z.]

3. *Toxic tetanus* is generally a consequence of strychnine poisoning; this is quite frequent in the dog. Among the principal tetanizing poisons may be mentioned brucine, nicotine, picrotoxine, thebaïne, caffeine, and ergotine. The discovery of tetanine, tetanotoxine, and other alkaloids which are generated by the bacillus of Nicolaïer establishes an analogy between traumatic and toxic lockjaw.

Symptoms of tetanus in general. Tetanic spasm appears, as a rule, in the muscles of the neck and head; from these it extends to the neck and shoulders, trunk, and extremities (descending tetanus); at times it is localized at first upon the hind quarters and spreads forward (ascending tetanus). In the first form the symptoms at the start are trismus and stiffness of the head, which is extended upon the neck and shoulders; in the second we notice an interference with the movements of the hind legs and contraction of the muscles of the tail. Sometimes the initial phenomena are vague and undecided; at others the disease is announced from the onset by tonic contractions which are almost generalized. In traumatic tetanus the contraction starts frequently in the region of the injured organ. The following are the different tetanized muscular groups:

1. Contraction of the masticatory muscles is designated by the name of *trismus*. In those cases where the two masseters are completely contracted the inferior maxillary is no longer able to execute the slightest movement; then the prehension and mastication of food are difficult or quite impossible.

2. Contraction of the auricular causes straightening and a drawing closer of the ears; of the great posterior of the eye, causes a retraction of this organ within the orbit and a protrusion of the mem-

brana nictitans upon the eyeball; spasm of the small nasal muscles leads to a dilatation of the nostrils, and that of the upper lip retracts and enlarges the mouth. In the majority of cases the muscles of the tongue, larynx, and pharynx participate in the tetanic spasm.

3. Contraction of the cervical extensors causes a straightening of the head and produces "the stag's neck" (stag disease); the cervical muscles are distended and hard to the touch.

4. Spasm of the extensors of the dorsal column is marked by extreme rigidity of the former. Upon the back, loins, and croup these muscles are as hard as wood. In some cases the distended neck and shoulders form with the back and croup a horizontal line (*orthotonos*); more frequently the neck and shoulders are extended and carried high backward, and the dorso-lumbar column is slightly depressed (*opisthotonos*); rarely the spinal cord and shoulders are incurved on the side (*pleurosthotonos*). Concerning the back of the carp (*emprosthotonos*), which is quite common in man, we have never seen it in domestic animals. In the horse the tail, which is kept turned up, often projects in a direct line from the vertebral column.

5. Contraction of the muscles of the legs renders their flexion impossible; the patient remains standing in a particular position, with the four members spread like the feet of a turtle.

6. Tonic contraction of the abdominal muscles tightens the abdomen; this is tucked up (doe belly).

7. Spasm of the muscles of inspiration renders breathing more or less difficult. Hyperesthesia and reflex hyperexcitability are observed, which are marked by anxiety, fear, and permanent *over-excitement*. The most insignificant causes produce paroxysms. The noise made in shutting a door is sufficient to cause a sudden falling of the patient. The body is wet with perspiration, which is always very abundant in serious cases and at the approach of death. Benign cases may, however, progress without *sudation*. The temperature is subnormal or slightly increased; but when tetanus ends fatally hyperthermia appears at the last moments, the temperature rises quickly and remains elevated for some time after death; we may find 42°, 43°, or 45° C., or even higher. In a horse, Beyer observed, twenty-four hours before death, a temperature of 39.2°; an hour and a half before death, 40.2°; at the time of death, 44°, and fifty minutes later 45.4°! This high temperature is explained by the intensity and permanence of the muscular contrac-

tion; nevertheless, it is probable that in some cases it is only apparent and due to an irregular distribution of the general temperature. They explain the *post-mortem* elevation by the heat which becomes free at the moment cadaveric rigidity is produced (coagulation of the myosine). The pulse, which is normal at the outset, continues unchanged when the disease is benign in character. In the horse, during the last few days, we may count 70 to 90 beats per minute; the circulation is accelerated when the patients are in a recumbent position. The arterial wall being the seat of spasmodic tension, the pulse is often found hard and small; in some cases, however, it is full, soft, and compressible. The respiratory movements are doubled from the beginning of the disease; at the same time spasm of the pectoral and abdominal muscles becomes accentuated and more and more accelerated; they also show considerable variations; the reflex increase of the intensity of the muscular spasm, which is occasioned by all kinds of excitement, produces this acceleration. An examination of the patient is sufficient to cause an augmentation of these movements, which may reach twice or three times the normal figure, and may be quadrupled even when the pulse is not sensibly accelerated. In the horse, during this period, from 80 to 100 respirations per minute may be counted. In fatal cases a progressive acceleration of the respiration is the rule, on account of the fixity of the thoracic walls, in that position which they have toward the end of the inspiratory act, and also because of the obstruction to the movements of the diaphragm, due to spasm of the abdominal walls; breathing can, therefore, no longer be performed without embarrassment. We observe also cyanosis or catarrh of the pituitary membrane, cough, and laryngeal, tracheal, and bronchial râles. When the disease terminates in death we may recognize symptoms of pulmonary hyperemia, œdema of the lungs, hypostatic pneumonia, and traumatic pneumonia.

In the digestive apparatus we observe from the first an interference with the prehension of food; this is often impossible even when the appetite is retained. Mastication is laborious and produces a peculiar liquid bruit; spasm of the pharyngeal muscles renders swallowing impossible; the boli accumulate in the buccal cavity and become partially decomposed or are ejected by the mouth and nasal cavities. Drinks and slops are taken much more easily than dry food; but frequently the animals play with the

drink which is given to them; they plunge their heads into the pail and make fruitless efforts to satisfy their thirst.

Peristaltic movements are generally suppressed; in some instances defecation is suspended, at other times the contraction of the muscular walls of the abdomen produces slight evacuations. Micturition is rare, and in some cases (spasm of the vesical sphincter) there is complete retention; the urine increases in density, and sometimes contains albumin; its reaction is ordinarily normal, but in herbivorous animals, in cases where inappetence is complete and when the disease develops rapidly, it becomes acid. A prolonged contraction has no influence upon the reaction of the urine or on albuminuria. In the acid urine of tetanic horses Friedberger has constantly found numerous oxalate crystals. Finally, in some cases there are frequent erections in *entire* horses and in geldings.

The intellectual faculties are generally preserved to the end of life; but the patients have an expression indicating fright, anxiety, and agony; their condition always inspires pity from persons coming near them and the veterinarian having them under treatment.

Symptoms of tetanus in the different species. I. IN THE HORSE. During this condition the head is carried high and extended; neck and shoulders are stiff, elevated, or even thrown backward; the ears are straightened and brought closer together, resembling hares' ears; the nostrils are much dilated and the eyes retracted, the *membrana nictitans* is prominent on the ocular globe, the pupil is dilated. The veins of the head are much distended; a viscous saliva drips from the mouth in large drops or long filaments; the tail is elevated, in some instances it is carried upon the median line, or deviates to one side or the other. The legs stand apart like those of an easel; the general attitude is one of immobility, the trunk is but slightly balanced upon the legs from time to time. The eyes indicate excitement and anxiety; the mucous membranes are injected; some muscular groups, principally the masseters and the muscles of the back of the neck, are hard and distinctly outlined, as if *carved*. The muscles of the jaws, neck and shoulders, back, lumbar region, *croup* and tail are as hard as wood. The tail and ears are elastic; when they are moved they immediately return to their former position. There is profuse sweating at the base of the ears, the lateral fascia of the neck and shoulders, the ribs and flanks; these are much tucked up. Respiration is accelerated and laborious. When approaching the patient

in order to examine it its excitement increases instantly; the lower jaw is pressed hard against the upper, and cannot be separated from it, even by exerting violent traction upon it; on parting the lips a fetid liquid runs out of the mouth, which is more or less filled with particles of food; if the head is lifted, the whole membrana nictitans obscures the greater part of the eyeball. Backing is extremely difficult or even impossible; turning is also very laborious; the trunk, neck, and shoulders cannot be flexed; the extremities are as stiff as stilts. In walking the legs are carried in abduction.

II. In BOVINES the clinical appearances are often greatly modified by symptoms of an acute or chronic septic endometritis. The animals remain quiet, as if nailed to the ground; the legs are spread, the head and back of the neck are distended, the ears are carried backward, outward, and upward. The look is anxious and haggard; the eye is sunk in the orbit and partially covered by the membrana nictitans, the protrusion of which is more marked as the head is carried higher. The *muzzle* is dry and hot; the mouth contains a more or less abundant quantity of thick and fetid mucus; its mucous membrane is hot; oftentimes the lips are slightly retracted, at other times the mouth is closed spasmodically; the movements of the lower jaw are interfered with. The muscles, especially the masseters, are distended and as hard as wood; the tongue is immobile and hard, and is sometimes found fixed between the dental arches. The ventral walls frequently undergo a remarkable lateral retraction; they fall perpendicularly upon the line formed by the extremity of the transverse apophyses of the lumbar vertebræ. The peristaltic movements of the rumen being suppressed, a slight tympanites occurs after a certain number of days. Defecation and micturition are no longer performed. The movements are very laborious. Reflex excitability is generally less than in the horse. Toward the end of the disease respiration becomes very dyspnœic.

III. In the SHEEP the gait is laborious; the legs are much contracted, transformed into rigid pillars and kept apart; the tail is carried horizontally in the axis of the body or inclined laterally. The back of the neck is straightened or thrown back; the jaws are *closed violently*. Later the respiration becomes dyspnœic. We often find the animals in a recumbent position, the legs are spread apart and the cervical region is incurved backward. The other symptoms are similar to those which occur in the ox.

It is not possible now to give a good description of tetanus in lambs; it has often been confounded with other diseases, mainly with pyemic arthritis.

Manifestations of tetanus in the *goat* resemble those seen in the sheep.

IV. In the *DOG* generalized tetanus seems to be very rare. A *statistic* of Möller gives a single case of generalized tetanus in 50,000 patients. In 25,000 sick dogs which we have observed we have never met it; but our observations establish that trismus is quite frequent in young animals. Generalized tetanus is marked by stiffness and difficulty in walking and extension of the head and cervical region; the ears are straightened, rigid, drawn together, and sometimes retracted; the eye is fixed and haggard, and the membrana nictitans appears upon the eyeball; the skin of the forehead is wrinkled; there is trismus; the animals can no longer bark; the muscles of the back of the neck and shoulders are hard, distended, and contracted (stag's neck); the legs are spread like the feet of a trestle; in many cases the tail is stiff; orthotonos or opisthotonos may be observed; the reflex excitability is increased; touching produces paroxysms.

V. In the *PIG* the symptoms of tetanus differ but little from those observed in other animals. The spasm begins generally in the muscles of the jaw, and extends rapidly to all the muscular groups.

Pathological anatomy. Anatomo-pathological observations are ordinarily negative. The lesions which are found are related to consecutive diseases or to accidental complications. In the nervous centres we find hyperemia, ecchymoses, softening, œdema, and transuded liquid, etc., all of which alterations may be wanting or are determined by complications.¹ In traumatic tetanus we sometimes observe in the nerves which originate from the wounded region a pink congestion of the sheaths, small hemorrhages, tume-

¹ Some authors claim to have succeeded in transmitting tetanus by injecting the cerebro-spinal fluid. With nervous matter obtained from the *bulb*, Nocard succeeded in inoculating a guinea-pig with chronic tetanus, which continued nearly six weeks and ended in recovery (see *Recueil Vét.*, 1887). By inoculating rabbits with the cerebro-spinal fluid of a man who was trephined during the course of tetanus, Dor produced instantaneous death without witnessing any tetanic phenomena. After the death of the trephined patient search for the bacillus in the cerebro-spinal fluid was unsuccessful. It was found only in a small hemorrhagic centre in the gray substance (see *Compt. rend. de la Soc. de biol.*, 1890).—N. D. T.

faction, and softening of the fasciculus (ascending neuritis). The blood is no longer decarbonized on exposure to the air; it is viscons, tar-like, imperfectly coagulated, and possesses a marked coloring power; numerous large ecchymoses and sero-bloody exudates are found under the serons and mucous membranes. The lungs are congested and œdematous, and may present hemorrhagic infarcts, splenization, a hypostatic pneumonia or a gangrenous lobular pneumonia may be found; here and there a vesicular and interstitial emphysema is met with. The heart is the seat of epicardiac and endocardiac hemorrhages, which occupy in preference the vascular furrows and pillars. Rigor mortis ensues rapidly, and quickly becomes pronounced. Sometimes the muscles are normal, at other times they are of a dark-red, yellowish- or bluish-brown shade; they may be studded with ecchymoses; in some cases they preserve their firmness; in others they are soft, as if cooked. In the muscular fibres the microscope shows an absence of the transverse striation, a granular tumefaction, and an irregular segmentation. The liver is commonly hypertrophied, yellowish, or yellowish-gray (infiltration and fatty degeneration). The spleen is tumefied, flabby, and filled with blood. The bladder is distended with urine and its mucous membrane studded with hemorrhages. The intestinal mucous membrane is ecchymosed.

Course. The course of tetanus is variable according to the species and individual. In the horse it commonly develops rapidly, and death takes place in one, two, or three days. This is a rule without exception. The disease is scarcely established before the patients are overcome by an intense trismus; all the muscles are contracted; we observe dyspnœa, a very marked reflex excitability, profuse sweats, a considerable acceleration of the pulse, and a high elevation of temperature; the patients fall, exhibit great agitation, and succumb in a few hours. In other cases death occurs more slowly, within four to eight days on an average. In others, again, when the spasm is moderate and of little extent, the disease may continue for several weeks. Sometimes, when the patients are apparently recovering, they are carried off by a complication (traumatic pneumonia). Kuhne gives the history of a horse that died on the fifty-seventh day in consequence of cavernous pneumonia.

Recovery before the third week is rare; about this time the spasm begins to decline, the appetite returns, respiration becomes

calmer, and the movements more free; very often recovery takes place only at the end of five or six weeks; there may be a continuance of the stiffness of movements, and tension of the extensors of the spinal column. Convalescence is long; it often lasts for months.

In the ox the course is less rapid than in the horse; the spasm is more slowly accentuated and never becomes so intense. There are cases where trismus is entirely developed only at the end of ten to fourteen days. The acute form, however, which is quickly fatal, is sometimes observed upon the ox. The duration of the disease rarely exceeds three weeks. Recovery never occurs before the fifteenth day.

In the sheep death usually takes place in from the sixth to the eighth day.

In the dog tetanus sometimes develops very rapidly (traumatic tetanus); in other cases, the patients linger from six to ten days. Recovery is announced toward the twelfth or fourteenth day.

Prognosis. The prognosis of tetanus is very grave. In the sheep and pig the mortality is about 100 per cent.; in the horse, from 80 to 85 per cent.; in the ox, from 70 to 80 per cent. In the dog the disease is less fatal. In newborn animals, especially lambs, recovery is rare. The traumatic form is generally considered more dangerous than the rheumatismal form. In the bovine species tetanus, which is consecutive to the puerperal state, is more serious than the rheumatismal and traumatic forms. The alarming prognostic symptoms are: a complete and rapidly-developed trismus (the majority of tetanic horses in which trismus is incomplete recover), rapid extension of the contractions to all muscles, intense dyspnœa, a sudden and considerable rise of temperature, quickness of pulse, falls upon the ground, and profuse sweating. In the horse the duration of the critical period is from fourteen to sixteen days; when this interval is passed we may hope for recovery.

Differential diagnosis. Tetanus may be confounded with cerebro-spinal meningitis, rheumatism, eclampsia, catalepsy, hydrophobia, pyemic arthritis (lambs, colts), and certain spasmodic attacks in newborn animals.

When tetanus is characterized by contractions of different muscular groups, the perfect preservation of cerebral faculties, absence of high temperature, and an external particular habit which it gives the patients, its diagnosis is easy. There may be difficulty in dif-

ferentiating the toxic tetanus of strychnine poisoning. The extreme rarity of tetanus in the dog, on the one hand, and the sudden appearance of the symptoms, the rapidity of the course, and the marked reflex excitability present in poisoning by strychnine, on the other hand, are usually sufficient to establish a diagnosis.

Treatment. A dietetic regimen should be begun, and the patients must be kept free from all kinds of excitement. Such are the two main indications of the treatment. It is advisable to place the tetanic subjects in spacious isolated stables, the floor of which is soft or covered with an abundant litter of short or hacked straw, in order that the animals shall be free from restraint or annoyance. We have often obtained good results from suspensory apparatus; the animals soon become accustomed to it. Suspension prevents falling, which may happen during the night especially, and produce an aggravation of the tetanic phenomena.

We should give the patients green fodder, slops, mealy drinks, liquid food and cold water *ad libitum*. With the object of preventing decomposition of the food, the rations should be small. When the animals sweat it is advisable to change their blankets frequently. Some practitioners have advised rubbing with straw when the body is in a perspiring condition; this is a harmful practice, on account of the excitement which it occasions. From time to time it is advisable to empty the rectum and produce micturition by careful rectal pressure or by the use of the catheter in females. In small animals clysters should be used.

Medicinal agents are of secondary importance in the treatment of lockjaw.

We may use narcotics: injections of hydrate of chloral (50 to 100 grammes per day, to be administered in a mucilaginous decoction in doses of 10 grammes per hour), subcutaneous injections of morphine (horse, 0.4 to 0.6 gramme of hydrochlorate of morphine), inhalations of chloroform (chloroform 1 part, ether 2 and 3 parts, until narcosis commences), and rectal injections of ether (25 to 50 grammes *pro dosi*).

The administration of medicines by the mouth is not practicable by reason of trismus, and is contra-indicated on account of the danger of entering the trachea.

In cases of traumatic tetanus the infected wounds require special care (disinfection, extraction of foreign bodies).

In large animals it is sometimes very difficult to treat wounds

without causing the animals to fall; in wounds of the foot it is often impossible to lift it. When the nature of the traumatism requires local treatment it may be necessary to anæsthetize the patient with chloroform. Poinot is said to have obtained good results by general frictions with carbonated water (10 per cent.) (?).

At the present time nothing positive is known as to the curative value of neurotomy in cases of traumatic tetanus. According to Rosco, plantar neurotomy has produced a rapid improvement and cure in a case of tetanus caused by a street nail.

A large number of drugs have been employed in the treatment of tetanus. Among narcotics, opium, morphine, ether, chloroform, hydrate of chloral, Indian hemp, bromide of potassium, hydrocyanic acid, cyanide of potassium, belladonna, veratrine, aconite, curare, camphor, essence of turpentine, tobacco, and even strychnine have been used. Cutaneous irritants (from laurel oil to cantharides ointment and fire), setons and trochisci, purgatives, copious bleeding, damp heat in the shape of vapor baths, washing, etc., and castration in the horse, have been tried. According to the authors who have recommended them, all these remedies proved of advantage. But, considered in the light of rational therapeutics, it is evident that many of them are liable to produce injurious effects and that others have no effect. There are no drugs the efficacy of which has been clearly demonstrated. [See page 165.]

EPILEPSY.

Nature. This is a relatively rare disease, although it is observed in all domestic animals, including the gallinacæ. It is a chronic cerebral affection, which is characterized by cerebral symptoms, sensitiveness, and tonic or clonic convulsions. It manifests itself in the form of attacks separated by intervals of greater or less extent, during which the patients present all the appearances of health. Recent experiments made upon animals have thrown some light on the nature and localization of this disease. At the present time two principal theories are in favor. The older, which considers the medulla oblongata and pons Varolii as the seat of this neurosis, has been sustained by the results of Nothnagel's researches. In his experiments on animals this author found in the pons Varolii a *centre of contraction*, the direct or reflex stimulation of which generated epileptic attacks, while irritation of the vasomotor centres

of the medulla oblongata led to a generalized cerebral anemia and loss of consciousness. But at the present time this doctrine is almost abandoned, and the external cerebral layer is generally considered as the seat of epilepsy. The stimulation of the motor centres of the cortical substance of the brain determines epileptiform spasms which are localized in the muscular groups the activity of which is governed by these centres; after the removal of the latter encephalic excitement does not produce any spasmodic reaction. In man we quite frequently observe epileptiform attacks in lesions involving the cortical substance of the brain. Von Bergmann has established this in all encephalic traumatisms; those which especially injure the cortical layer of the brain most frequently lead to epileptiform attacks—true epileptics. The constant coexistence of spasms and loss of consciousness points to the localization of epilepsy in the external encephalic layer.

The experiments of Hitzig, Fritzsche, and Luccani have shown that electric irritation of any region of the external layer of the brain may generate epileptiform attacks; but these are occasioned by the slightest influences acting upon the motor centres, and a destruction of the cortical substance, with the exception of these regions, does not prevent their manifestation. In order to be effective, the stimulation which provokes the attack must be transmitted to the motor centres through the gray substance. Such are the principal facts upon which is based the doctrine which connects epilepsy with functional troubles of the motor centres of the external cerebral layer.

The modifications which are undergone by the nervous substance are unknown. Cerebral anemia which is suddenly produced by vascular spasm was formerly considered as the cause of these attacks (Küssmaul, Tennor), but most investigators have been unable to find this anemia. The exciting cause of epilepsy seems to consist of a passive state of excitement of the cortical layer of the brain and motor centres, a phenomenon which may be due to an abnormal chemical or molecular composition of the mucous substance (Rosenbach). In certain benign cases epilepsy in man is marked exclusively by a loss of consciousness (here encephalic excitement does not act upon the motor centres of the cerebral envelope).

Etiology. From a practical point of view, several varieties of epilepsy may be recognized :

1. *True, primitive, idiopathic, and spontaneous epilepsy.* This is

a purely functional neurosis of the encephalon, in which the latter is not the seat of any important anatomical lesion. It is generally attributed to an abnormal irritability of the cortical substance of the brain, but its nature is unknown. It is observed at all ages and in animals of both sexes. Among its predisposing causes heredity plays a leading rôle in domestic animals. In two herds and in animals which were procreated by two epileptic bulls, La Notte recognized numerous cases of epilepsy; in cows the disease ordinarily appears after the first calving; in oxen the initial attacks were seen after the first performance of work. Therefore, in studs epilepsy is considered, with good reason, to be an hereditary disease. It is possible that certain impairments of general nutrition—rhachitis, for instance (a case observed in the dog by Siedamgrotzky)—and the delicate condition of young animals create a predisposition to epilepsy. According to Strube, breeding stallions are particularly subject to it.

The causes which are capable of determining the attacks are, as in man, mental excitements, fear, irritation, and fright. Romer mentions the case of a gelding, fourteen years old, always belonging to the one owner, in which the first attack was occasioned by a white sheet thrown in front of him when he was hitched to a plow. La Notte has seen the spasm occur in a horse which was frightened by a rocket that suddenly began its flight in the neighborhood. Leidesdorf saw it in a dog which had been scared by a locomotive. In the horse the attack is frequently occasioned by very intense rays of light, or by passing from a strong light into a comparatively dim one; this form of the disease has also been attributed to rapid races in the direction of the setting sun, and upon roads crossed by the shadows of trees, luminous rays reflected upon the surface of water or snow, etc.

2. *Symptomatic epilepsy.* This is due to certain anatomical alterations of the brain or of its envelopes; such as neoformations, parasites (cysticercus in the pig), abscesses, old encephalitic lesions, tubercles, chronic pachymeningitis with thickening of the dura mater, exostosis, etc. This form of epilepsy consequently differs essentially, as to its nature, from true epilepsy; its symptoms only are the same; thus the spasms which are produced by it are designated by the name of *epileptiform* attacks. An autopsy gives precise information as to the causation of this affection.

3. *Traumatic epilepsy.* This has its starting-point in wounds of

the cranial wall and brain. Its pathology varies according to the case. It may be the expression of a lesion of the cortical cerebral envelope, of its compression by blood, by bony spicula or alterations of the meninges. The spasms sometimes occur long after the action of traumatism. This form of the disease has been produced experimentally by blows upon the cranium (Westphal); it appeared a few weeks after the traumatism. Lucanni claims to have observed in a dog the hereditary transmission of epilepsy which was consecutive to mutilations of the cortical substance of the brain.

4. *Reflex epilepsy.* This is observed in certain nervous and predisposed animals under the influence of irritations which act upon the peripheric nerves. Among these irritations we may mention wounds and contusions, compression of the nerves by tumors, irritation of the mucous membranes by parasites (*Ascaris megalocephala* in the horse; tenias and *Linguatula tenioides* in the dog). In nervous dogs constipation may determine epileptic attacks. In the horse irritation of the buccal mucous membrane by wolf's teeth sometimes produces them during eating. Gerlach saw a horse overcome by an attack and fall the instant he was touched upon his withers, which had become very sensitive. In the rabbit Brown-Séquard produced experimentally this form of epilepsy by a traumatic lesion of the peripheric nerves; a section of the sciatic determined epilepsy within from eleven to seventy-one days; the disease persisted for quite a long time. Its transmission by heredity has several times been recognized.

Pathological anatomy. In true epilepsy no alteration of the cerebral substance exists. In the other forms we meet with tumors, exostoses of the cranial wall, thickening, adhesions or ossification of the meninges, encephalitic centres, chronic hydrocephalus, cerebral œdema, partial cerebral atrophy, etc. In an epileptic cow, Voigtländer recognized a suppurating encephalitis of the right hemisphere, which was consecutive to a fracture of the horn. In a dog Siedamgrotzky found asymmetric and irregular asperities upon the inner fascia of the bones of the skull; the capacity of the cranial cavity was much lessened, the brain was compressed and shrunk, and chronic hydrocephalus was present; the ventricles were dilated and the neighboring cerebral regions atrophied.

Symptoms. Epileptic attacks are sometimes characterized by generalized convulsions, accompanied by falling and loss of con-

sciousness (general or complete epilepsy of authors); at other times by spasms localized in certain muscular groups, especially those of the head, neck, and shoulders (incomplete, partial epilepsy). Between these two forms we may observe in the same animal all possible transitory states.

1. *Serious epileptic attacks* (complete general epilepsy), such as we may observe in animals in the stable or during work, appear sometimes abruptly, without any precursory phenomenon; the animals stagger for a moment, the dog and pig often utter a piercing cry; all fall as if crushed, and are seized with tonic and clonic spasms. But generally we observe prodromes which are very similar to vertigo. In rare cases the attack is announced by a sensitive *aura*; by particular troubles of sensitiveness which occur in certain limited regions of the body. Hertwig has seen two horses which immediately after the crisis tried to scratch their heads with the right hind leg. This is the ordinary course of the attack. If the horse is tied fast, he will go backward suddenly and pull on the tether; when at work he will stop, over-excited and anxious. The dog wanders about timidly, as if he had lost consciousness, or he looks around him in an anxious manner and shakes the head. In all species the eye is prominent, fixed, and haggard. The animal trembles, staggers, backs a few steps, or spreads its legs. The convulsive contractions generally begin in the head or neck and shoulders; the eyelids quiver and the eye rolls in its orbit; we observe strabismus, convulsive contractions of the muscles of the lip, nostrils, cheeks, ears, and abnormal movements of the jaws, which render the saliva foamy. Sometimes the lower jaw is opened and closed convulsively, at other times it is tightly closed, drawn against the upper; grinding of the teeth is common. By degrees the spasms spread to the trunk; the head and neck are violently moved from one side to the other, sometimes as far as the pectoral wall. The patient loses its equilibrium and falls; clonic contractions quickly reach their maximum of intensity and are propagated to the extremities; the legs are the seat of very violent convulsive movements. Intelligence and sensitiveness are entirely abolished.

The mucous membranes of the head, especially the conjunctiva, are pale at first; the pupils are dilated and insensible to light; reflex movements are suppressed. The heart is irregular and slow—the beats are less than half of the normal number; the pulse is small and hard. Respiration is laborious, rattling, and dyspnoeic

during decubitus. The dog and pig utter piercing cries, similar to those of young animals. Consecutively to dyspnoea and violent muscular contractions, there occurs toward the end of the attack a marked venous congestion of the head, and at the same time we observe an abundant sudation in the neighborhood of the elbows and flauks.

Sometimes the convulsions cease abruptly, at other times they disappear gradually in order to make place for a contraction of short duration, which is accompanied by micturition and involuntary defecation. As soon as the attack has spent its force consciousness and sensitiveness reappear rapidly, the animals regain their feet, and by degrees return to their normal state; but symptoms of weakness and exhaustion persist for some time. When the attack is over the dog regains his feet, shakes himself, and looks around in a frightened way; in some cases, however, he is overcome by a deep sleep, which is marked by rattling. As an immediate consecutive phenomeuon, some authors mention also an extraordinary maniacal excitement, in which the dog wanders around without aim and tries to bite. Schrader observed a horse which bit the right front leg as soon as the paroxysm was over.

2. In *benign epileptic attacks* (incomplete, partial epilepsy), which are generally accompanied by loss of consciousness, the spasms sometimes remain localized on certain muscular groups of the head (working of eyelids, strabismus, turning of the eye in its orbit, spasmodic convulsions of the muscles of the face and lips). In the horse benign attacks show a great analogy to vertigo; they are observed under very different circumstances, but especially when working, and in subjects wearing a bridle which is fitted with blinds. When the spasms become more violent we observe convulsions of the head, neck, and shoulders, and trembling of the front quarters; the animals lean against neighboring objects or stumble or fall on their knees.

The duration of the spasm is variable, but in general it is proportional to its intensity. It varies between a few minutes and half an hour (we have observed a case where it lasted only fifteen seconds). The attacks occur at very irregular periods; they may be repeated several times on the same day; in some cases they are not observed for months or years; in some instances they are more frequent in the beginning, and in others toward the end of the disease. The exciting causes have been described when

treating of the etiology. We ought to add, that in domestic animals as well as in man the touching of certain parts of the body (epileptogenic zones) may determine an attack. Schrader has reported the history of a horse in which it sufficed to touch the middle of the left fascia of the neck and shoulders in order to cause a seizure. The violence of the attacks is very variable even in the same animal. In the horse benign attacks often remain stationary for many years as far as their degree of intensity is concerned. During the periods which separate these the animals show all the symptoms of health ; mental depression, cerebral troubles, dulness of the senses and intelligence are rare. If the trained dog *forgets its tricks*, that may be due to chronic hydrocephalus or to cerebral atrophy, which often coexist with epilepsy.

The course of epilepsy is essentially chronic. The disease persists for years, sometimes through life. It is very exceptional to see it disappear after a few months, and when this happens it is very likely that it has been confounded with some other affection. It does not cause death directly, but, in falling, the patients are exposed to serious wounds and cerebral hemorrhage.

Differential diagnosis. The diagnosis of epilepsy is based upon its chronic course and upon the appearance of spasmodic muscular attacks, which are accompanied by stupefaction. We may confound with epilepsy :

1. *Eclampsia and acute cerebral spasms.* These latter are epileptiform or epileptoid attacks which occur in the course of various acute diseases of the brain, in infectious diseases (dog diseases), and in certain intoxications, or are produced as ordinary reflex symptoms (dental spasms, etc.). They have a close analogy with epileptic attacks ; like the latter, they are accompanied by mental symptoms. They are characterized by an acute development, but this is not constant ; besides, the reflex cerebral spasms may become prolonged when the exciting cause acts for some time (intestinal parasites). In the latter case, a distinction is very difficult and the *intra-vitam* diagnosis is frequently impossible.

2. *Vertigo*, which may simulate benign epileptiform attacks.

The differentiation of these two diseases is based upon the absence of convulsions in vertigo ; but in mild epileptiform attacks convulsions are not marked (in man they may be wanting entirely), and the movements of the legs—causing falls in attacks of vertigo—are similar to those caused by convulsive contractions. It is there-

fore not possible scientifically to draw a precise line between epilepsy and vertigo.

3. *Thrombosis* of the terminal branches of the posterior aorta (iliac, lumbar, and crural arteries), causing a falling of the hind quarters, has sometimes been mistaken for epilepsy. The diagnostic symptoms are: the conditions found on rectal exploration, a peculiar paresis of the back legs, and the power to produce at will the characteristic symptoms of this disease by exercising the animals or by compelling them to make muscular exertions.

In judicial veterinary medicine the diagnosis of epilepsy is very important. In some countries, in fact, this disease is considered a sufficient cause for setting aside a contract of sale if its existence is discovered within a certain time (the limit allowed in Würtemberg, Baden, and Hesse is twenty-eight days; Bavaria, forty days). A delicate question is, whether *reflex epilepsy* presents the characters of a disease sufficiently marked to annul a contract of sale. We are inclined to answer negatively when we reflect that there are isolated cases of this form of epilepsy in which recovery occurs when the causal peripheric irritation is suppressed (expulsion of intestinal worms, planing of teeth, etc.). These cases must be considered as examples of acute cerebral spasms of longer or shorter duration; they possess no features warranting the setting aside of a sale; but this is far from being so in all cases of reflex epilepsy. We may also remark that in general we do not succeed in discovering the seat and nature of the exciting cause. From a clinical point of view we are almost always obliged to be satisfied with a diagnosis of "epilepsy."

Certain medicinal agents are of value in the treatment of true epilepsy. When a cure is thus obtained we are unable to distinguish this form of reflex epilepsy. The production of reflex epileptic accidents implies, besides, the existence of an intervening nervous state as a predisposing influence; peripheric excitements, in fact, determine attacks in only a majority of cases. A similar condition of the nervous system simulates epilepsy (in man heredity has been recognized as a causative agent in accidental epilepsy, which is developed in predisposed subjects, as well as in true epilepsy); at all events it lessens the value of animals, for in those liable any great excitement may produce an attack.

Experiments upon animals have demonstrated that true hereditary epilepsy may be developed under the influence of peripheric

irritations. Quite recently Von Bergmann has advocated a theory¹ according to which epileptic phenomena produced by an injury to the peripheric nerves must be regarded as true epilepsy.

For these reasons we think there is no occasion in legal medicine to distinguish reflex epilepsy from true epilepsy, and that the former ought to be considered a valid reason for setting aside a contract of sale every time it is possible to recognize it.

Treatment. Prophylaxis indicates the removal of all exciting causes. During an attack it is advisable to hold the head fast, to cover the eyes, and to restore the deviated head and neck to their normal position; by these means we sometimes succeed in subduing the spasm, and in many instances falling is prevented. In order to shorten the duration of the attacks inhalations of aromatic substances or of chloroform and douches of cold water have been advised; but in large animals we cannot well make use of these agents, and in small species they are almost without effect.

The disease itself has been combated by a number of remedies which act by diminishing the cerebral excitability. In the dog the treatment offers some chances of success; in the other animals the disease is too rare to permit a test of the efficacy of the various medicinal agents recommended. Our personal observations are in accord with those of many others in establishing that bromide of potassium administered for a sufficient length of time is particularly beneficial, especially in the dog; this animal may be given a watery solution of this drug three or four times daily in doses of 0.50 to 1 gramme. We may prescribe: bromide of potassium, 30 grammes; distilled water, 300 grammes, of which solution a tablespoonful or teaspoonful is to be administered three or four times daily (preparations in which a certain proportion of sugar enters rapidly decompose). In the horse and ox we give iodine associated with other salts or mixed with aromatic plant powder (20 to 50 grammes). Among the agents which are employed we may also mention nitrate of silver (dog, 0.02 to 0.1 gramme per day; horse, 1 to 2 grammes); oxide of zinc (dog, 0.05 to 0.2 gramme); belladonna (sulphate of atropine, 0.005 to 0.02 gramme for the dog, 0.05 to 0.1 gramme for the horse), ammoniacal sulphate of copper, and valerian root.

The employment of epileptic horses is attended with certain dangers. Such animals ought to be excluded from public service.

¹ Von Bergmann: Sitzung der militärärztl. Gesellschaft zu Berlin, von April 21, 1887.

ECLAMPSIA.

Generalities. In veterinary and human medicine for a long time there has been designated under the name eclampsia a group of morbid states of very different gravity and nature. If taken in its widest acceptation, the word eclampsia is applied to several convulsive states having as a common manifestation tonic or clonic contractions of acute development, which are accompanied or not by mental troubles, and terminate sometimes in death, at other times in cure. This very comprehensive definition is applied to a series of spasms which are purely symptomatic; they are related to well-known alterations, but do not characterize anatomically any true or independent disease: such are acute cerebral spasms which appear during the course of diseases of the encephalon, epileptiform accidents of disease of young age, symptomatic convulsions of uremia or of certain poisonings [for some time known as uremic eclampsia, saturnina (lead-poisoning), etc.].

At the present time the expression, eclampsia, has a more restricted signification; it comprises epileptiform spasms produced independently of any organic alteration, which consequently exist in a condition of pure neurosis. This idea associates eclampsia with "acute epilepsy;" their only differential characters would be a rapid development of the first and the likelihood of the other to return. Eclamptic spasm is produced by a reflex in the same way as epileptic spasm. It is sufficient to examine the scattered observations on certain spasmodic conditions found in our publications under the name of eclampsia in order to be convinced that this neurosis, considered in its form similar to acute epilepsy, exists in animals. This neuropathic type comprises *convulsions of newborn* and young animals (*eclampsia infantum*), as well as certain cases of *acute reflex spasms* which have been observed in adult animals.

Eclampsia of nursing female dogs, in which the mental faculties are unimpaired, is quite different. We have nevertheless related it to acute eclampsia on account of the obscurity which still surrounds its nature.

In the chapter on *parturient fever* we have shown that the name *eclampsia* is not a proper one for this trouble.

I. ECLAMPSIA OF YOUNG ANIMALS is especially observed in the dog (at the time of cutting teeth) and in young pigs (Hering). It is

expressed by convulsions which are similar to those of the eclampsia of children, by a turning of the eyeball in the orbit, trembling, spasms of the masseter muscles, grinding of teeth, ptialism with discharge of foamy saliva, and convulsions in various parts of the body; there are some which succumb uttering a piercing cry. Its cause resides sometimes in an abnormal nervous disposition, at other times the peripheric irritations (cutting of teeth, helminthiasis, gastric or intestinal catarrh, etc.). Its treatment is the same as that of epilepsy; it consists in the use of antispasmodics; bromide of potassium, hydrate of chloral, etc. We may also use a decoction of valerian root in clysters.¹

II. ECLAMPSIA OF ADULT ANIMALS is rare in a general way. In a majority of cases its manifestations are identical with those of acute reflex epilepsy. There are subjects in which attacks are occasioned by simply touching certain injured regions the integument of which is very sensitive (skin at the base of the horns). Epileptiform accidents which are determined, in the dog, by acari in the external auditory canal (Guzzoni, Vocard, Mègnin), must be related to eclampsia, and a large number of cases of reflex epilepsy appear to be repeated attacks of eclampsia; this, besides, may be transformed into true epilepsy. It is impossible to understand its exact cause. Lustig has recognized epileptiform attacks in a convalescent horse which had suffered from a serious disease of the lungs. Albrecht has observed it several times in cows shortly after calving; the phenomena mentioned by this author had nothing in common with those of parturient fever; they consisted of attacks of spasmodic extension of the back of the neck and head, which were accompanied by grinding of teeth, loss of intelligence, falling, convulsive movements of the legs, turning of the eye in its orbit, etc. The continuance of these attacks varied from a few minutes to half an hour; during the intervals the animals seemed to have entirely recovered. The minimum duration of the disease has been two days, the maximum eighteen days. Similar seizures have been described in the goat. In this form of eclampsia the spasms are undoubtedly occasioned by an irritating condition of which the uterus is the seat.

III. ECLAMPSIA OF NURSING FEMALE DOGS is as yet imperfectly known from an etiological and pathological point of view. It is

¹ In young dogs I have often seen these conditions due to the presence of intestinal worms, which quickly yielded to the action of vermifuges.—L. T.

only observed during the nursing period. It is possibly due to an irritation which begins in the nervous ramifications of the mammary glands; but here, again, we must admit a certain predisposition to spasms. Delicate, intelligent female dogs which are well bred, and those in which lacteal secretion is abundant, are most frequently affected. As occasional causes they mention especially mental troubles (loss of progeny, fright, fear of the whip) and cold.

The symptoms appear from the second to the thirtieth day after parturition. They consist essentially in attacks of tonic or clonic spasm which preferably affect the extensor muscles; the mental condition is normal. The attack appears suddenly without prodromes. We remark a certain restlessness, excitement, and anxiety which are especially expressed by the look; breathing is accelerated, short, laborious; the circulation is also accelerated, the mucous membranes of the head are injected, the hind quarters are affected by a rapidly-increasing paresis. Soon the animals, having become unable to stand up, fall and stretch themselves upon the ground (costal decubitus); the legs are stiff and contracted. When the patient is placed upright he remains immobile; the extremities are spread like the feet of a trestle. All voluntary movements are impossible. From time to time we observe a convulsive trembling of the legs and trunk; then clonic spasms appear, which are generally violent and affect the extensor muscles of the legs, neck, and back, more rarely the temporal and the eye muscles. With the appearance of spasms the respiration is more and more accelerated; it becomes dyspnoic, rattling (we may count as many as 100 respirations per minute); the mouth is wide open and foaming; the tongue hangs out of the mouth. The heart's action becomes tumultuous; we may count 160 or more pulsations per minute. The mucous membranes of the head become more and more congested; we may recognize hemorrhages in it, especially on the conjunctiva. The jaws are opened and closed convulsively, the tongue is drawn back, we observe frequent efforts at swallowing (Mauri). Prehension, micturition, and defecation are impossible. In most subjects the abdomen is sensitive to palpation. The urine which is ejected after the attack is said to be albuminous (Benjamin). Often, in the beginning, the teats are tumefied, hot, and full of milk; later they are found to be flabby and empty. The intelligence is not troubled, and sensitiveness is intact. As Mauri very judiciously remarks, the physiognomy expresses much more anxiety than pain. The

pupils have preserved their normal dimensions. Various external irritations do not aggravate these symptoms to any extent.

Course. The course of eclampsia is rapid. The attacks generally last for one or two days; they may, however, disappear after a few hours. The intensity of the spasms is not uniform; we observe remissions during which the animals try to take a standing position, and succeed quite frequently in raising themselves upon the hind quarters. The disappearance of spasms gives place to a sleepy condition. When the disease is left to itself death soon results; it occurs twenty-four to forty-eight hours after cerebral paralysis or asphyxia. A rational treatment leads to recovery within a relatively short time—often in one hour. We have observed cases of spontaneous cure. We should not confound eclampsia of nursing female dogs with poisoning by strychnine. In the former we do not observe hyperæsthesia, which is so characteristic of strychnine poisoning.

Treatment. The treatment consists in making either subcutaneous injections of hydrochlorate of morphine (Siedamgrotzky) in a dose of 0.02 gramme in a solution of 1 gramme of distilled water, or inhalations of chloroform, which are to be continued till narcosis commences.

Urethane (5 to 20 grammes) and hyponone (0.25 to 2 grammes) have also given us good results. Mauri has obtained favorable effects with hydrate of chloral (0.5 to 2 grammes). Zündel has advised syrup of chloroform (chloroform, 1 gramme; syrup, 100 grammes; to be administered per half teaspoonful at intervals of two hours).

The efficiency of these narcotic and vaso-dilator agents permits the formulation of a theory of the nature of the disease: we may admit that this consists in a circulatory trouble of certain *spasm centres*, a trouble which would appear suddenly in order to disappear quite as quickly. Some observations which have been collected by Hertwig aid in the support of this hypothesis. The spasms are undoubtedly the consequence of anemia of certain cerebral centres, and are produced by reflex excitement starting from the nervous mammary ramifications. We have, in fact, observed several times, by means of the ophthalmoscope, a pronounced anemia of the optic papilla. The autopsy does not reveal any anatomical alteration of the encephalon.

The salutary action of chloroform—antidote to all spasmodic

poisons—would be explained by admitting as an inducing cause of eclampsia a toxic agent—a ptomatotetanine.

On account of a want of positive facts bearing upon the etiology and pathology of eclampsia the field is, as can be seen, widely open to conjectures.

CATALEPSY.

Nature. Catalepsy is a particular, periodic morbid state, in which the whole muscular apparatus is “in a state of constant contraction,” while the passive movements and displacement of certain regions are performed with unexpected ease. Intelligence and general sensitiveness are much affected. According to some authors, the cataleptic rigidity is the result of a momentary augmentation of the normal tonicity of the muscles of animal life; others consider it the consequence of an abnormal conducting resistance of motor centres and of a diminution of excitability of the muscles by the will and external irritations.

Catalepsy is observed in animals either as an independent and primary affection, or as a secondary phenomenon, which appears during the course of other nervous diseases (cataleptic attacks). Landel has mentioned a case of secondary catalepsy in the ox. In general, primary catalepsy appears to be very rare in animals. It has been recognized in the dog, horse (Hering), and the prairie wolf (Leisering). (In the human species, where it is much more common than in animals, it is considered as a form of hysteria.)

Etiology. The causes of catalepsy are unknown. It has been attributed to cerebral emotions (fright, fear, etc.), but these appear only as a determining condition of the attacks. The etiology is dominated by an uncertain nervous predisposition. The affection shows a great analogy with hysteria in the human species.

Pathological anatomy. Catalepsy, as well as eclampsia and tetanus, is not characterized anatomically by any alteration of the central organs. It must be regarded as a functional neurosis of the brain and spinal cord. Sometimes we find in the muscles certain secondary lesions (hemorrhages, turbid tumefaction, fatty degeneration; vitreous degeneration of the muscular fibres of the heart; hemorrhages of the stomach and intestine, etc.) which are similar to those seen in tetanus (Fröhner).

Symptoms. They appear suddenly, without any precursory indications; in some cases, however, we observe anxiety, phenomena

of excitement, loss of appetite, etc. The rigidity starts in the muscles which are in an active condition at the time of the attack, and rapidly extends to the whole muscular system. The animals are unable to make the slightest voluntary movement; they remain immobile—petrified—in the position in which the cataleptic attack has surprised them. The affected muscles are distended, hard, and prominent under the skin; in the beginning they offer a certain resistance when we try to displace them, but gradually their rigidity is diminished, and later disappears entirely. Intelligence and sensitiveness are more or less affected. The eyes are fixed, dull; the pupil is sometimes dilated, in other instances very much contracted (reduced to the size of a pin's head). Sensitiveness of the skin and mucous membranes is diminished or completely abolished; the same is the case in the special senses (sight, hearing, smell). The urine which is passed after the attack is albuminous; in the dog it contains a large proportion of coloring-matter of the bile. The circulatory and respiratory apparatus does not present anything abnormal. In a cataleptic dog Fröhner observed a hypnotic state similar to somnambulism in man.

The attacks may be repeated; their duration is uncertain: according to Spinola, it varies from a few minutes to a few hours; Hertwig speaks of a duration of several weeks. In the dog Fröhner observed two cases which lasted twelve and thirty hours respectively. In general, the cataleptic state is not fatal. There are, however, cases where the animals perish from inanition (Hertwig). In the dog Fröhner has seen death occur on the seventh day; the cataleptic rigidity had given place to a relaxation of the whole muscular apparatus, comprising the muscular system of the intestine and bladder; the temperature had become gradually lower and the coma deeper.

Treatment. Narcotics appear to be indicated (morphine, bromide of potassium); little is known of their effects, but they must be considered very uncertain. We may try to bring back the reflex excitability by means of cold douches or electric currents.

CHOREA. ST. VITUS'S DANCE.

Nature. St. Vitus's dance in man is characterized by permanent convulsive movements of the muscles of animal life, which are accompanied by more or less marked disturbance of sensitiveness

and intelligence. These movements are always independent of the will; there are, however, cases where we observe an indication of design. The disease has borne the names of "muscular anxiety," "muscular delirium," and "twitching disease." Modern authors generally consider it as a cerebral affection. Among its causes they especially indicate capillary emboli of the corpus striatum and optic thalamus; in man, chorea frequently complicates articular rheumatism, endocarditis, and valvular alterations. The results obtained by Chauveau by section of the cord of choreic dogs establish that in the canine species the affection designated under the name of chorea is of medullary origin. The restricted number of observations related in veterinary medicine and the lack of clearness of some of the descriptions do not permit us to decide upon the similarity or relations which exist between the chorea of animals and that of man; nevertheless, it seems that, in the former, they have described under this name some cases of cerebral spasm or simple reflexes and benign eclamptic or epileptic attacks. Spasms which occur during the disease of young age in the dog (distemper) have no choreic character; they are but reflex clonic contractions.¹ But sometimes true chorea appears after the distemper in animals which are in full convalescence. It has been recognized in the horse, ox, dog, cat, and pig.

Etiology. The causes of chorea are unknown. It especially attacks young, weak, and anemic animals. A predisposition which is created by congenital or accidental influences of the nervous system appears to dominate its etiology.

Symptoms. Sometimes the characteristic convulsive contractions are localized in the muscles of the head and neck, in other instances they are generalized. In one horse Hering observed alternate movements of lowering and raising the head and an anterior member. In another U. Leblanc saw convulsive movements

¹ Clonic convulsions, which are observed so frequently in the dog, and are designated under the name *chorea*, are in fact *tics*, and cannot be likened to human chorea. This is essentially characterized by *irregular, contradictory, and arhythmic* movements, which are very different from rhythmic movements. Tics of the dog, which are improperly designated under the name of chorea, are indicated by spasmodic contractions which are localized on certain regions or more or less generalized, but which are of *uniform intensity* (or nearly so) and *perfectly rhythmic*. Gilbert, Roger, and Cadiot have established by experiments that tic of the face, in the dog is caused by functional disturbance of the origin of the seventh pair. (See Recueil Vét., 1890.) True chorea is very rare in all animal species, except the dog.—

in all the superficial organs: the foot, head, front quarters, hind quarters, ears, lips, eyelids, etc., were affected successively by clonic contractions. In a cow Anacker observed lateral rhythmic movements of the head and neck, while the hind legs exercised a *true dance*. In an ox Schleg noticed similar movements of the head and a greatly accentuated flexion of the front legs at the time of starting to walk. In young pigs Hess and Verwey have recognized rhythmic clonic spasms of all the muscles of the trunk; in some of these the head was constantly carried from one side to the other; in several the hind quarters were lifted from the ground at every spasmodic contraction.

Chorea of animals almost always runs a slow course and assumes a chronic type. In young pigs Hess has seen recovery occur within a period varying from seven weeks to several months. In a horse which was observed by U. Leblanc chorea persisted for more than one year.

The *prognosis* is relatively of little gravity.

Treatment. As in other neuroses, this consists in the use of anæsthetics and sedatives: chloral hydrate, bromide of potassium, etc. We may also use oxide of zinc or arsenious acid. Legros and Onimus have seen the attacks disappear in a dog after the rectal administration of 3.5 grammes of chloral hydrate. In this species Palombo has obtained cures by immersing the patients in cold water five or six times daily.

VERTIGO.

Nature. The word *vertigo* serves to designate abnormal sensations which are fugacious and periodical, and closely allied to the relations of the animal with the outer world. It refers to sensorial and motor perturbations, with loss of equilibrium or an impossibility for the animal to preserve a standing position. Sometimes these aberrations are due to functional troubles; at other times they depend on anatomical alterations which are developed in the cerebellum, which is the seat of co-ordination of movements. The former are rarest; in other words, vertigo is more frequently a symptom than a true neurosis. For practical reasons we have included it among nervous affections which are deprived of any anatomical criterion. Its study is important from a legal point of view, for in many countries it is included, with good reason,

among such diseases as set aside a contract of sale. But if considered from a clinical point of view, vertigo has only the value of a symptom, and belongs to the domain of general pathology.

AFFECTED ANIMALS. Vertigo is quite frequent in the horse; rare in the ox, dog, pig, and sheep. As a general rule, it affects adult or old and full-blooded horses, those that are well fed and perform but little hard work, and especially fine animals which spend the greater part of the day in the stable. It appears ordinarily in harnessed animals, more rarely in those that are mounted; it is hardly ever observed in horses while at rest. It is especially in the spring that many cases are recognized; a prolonged stay in the stable during the winter season, abrupt variations of temperature when the good season returns, and shedding seem to constitute the principal conditions of this frequency of vertigo in the spring. Certain horses which show regularly several attacks at this time are free from it during the rest of the year.

Etiology. 1. *Essential vertigo* is the result of influences which are absolutely unknown. It is not related to any anatomical alteration of the cerebellum. In veterinary medicine this form has been designated by the expression "cerebral vertigo."

2. The name *symptomatic vertigo* has been given to attacks having as a cause anatomic intra-cranial alterations: tumors (cholesteatomata in the horse—Vernaut, Cazalas, etc.), centres of encephalitic softening, chronic cranial meningitis, lesions of vessels of the brain, etc. Vertiginous attacks which are produced by acute encephalitis or by cerebral anemia consecutive to abundant hemorrhages, to the discharge of a large quantity of fluid accumulated in the splanchnic cavities, or to the escape of gas from the tympanic cavity, etc., do not deserve the name of vertigo, for this affection is essentially characterized by the recurrence of the attacks.

3. *Congestive vertigo*, which is due to circulatory troubles in the brain, is the most frequent type. It may be produced by various causes: diseases of the heart, of vessels, lungs—morbid states accompanying passive cerebral hyperemia, which lead to the same consequences as arterial anemia of the brain; determines the irritation of the cerebellum on account of the want of oxygen or by accumulation of carbonic acid in the blood; we observe, therefore, vertiginous attacks in the dog affected by valvular lesions, and in the horse during the course of degenerative chronic diseases

of the lungs, or in the case of aneurism of the pulmonary artery (Lustig), etc. Compression of the jugular veins by the collar, breast-strap or throat-strap, by an excessive checking or by too great tractions on the bridle, may also generate vertiginous attacks. The pathogenic influence of these various causes is favored by forced movements, great heat, hot and poorly-ventilated stables, plethora, etc. Anemia of long duration is frequently accompanied by vertiginous phenomena.

4. *Consensual or reflex vertigo*, which is often confounded with reflex epilepsy, is produced, like the latter, by transmission to the cerebellum of a peripheric excitement. *Optic vertigo* belongs to this group. It is quite frequent, and has as principal causes: blinds with a smooth and shining inner surface, very intense solar rays (lakes, rivers, and snow reflecting the rays of the sun), a sudden passing from shade to light, certain eye diseases, such as amaurosis in the early stage. Irritation of the external auditory canal by foreign bodies may also produce vertiginous attacks, the pathologic action of which, however, remains doubtful.¹

5. *Abdominal vertigo* is produced by certain diseases of the abdominal viscera (chronic intestinal catarrh, stagnation in the portal system, diseases of the liver).

6. *Physiological vertigo* is remarked in horses that are compelled to walk in a circle (mechanical ring action) and in those which are transported by boat or railroad. Hering claims that the seasickness of man is also observed in animals, especially in the dog, more rarely in the horse.

Certain poisonings (*Equisetum*, *Lolium tenulentum*, solanine, alcohol, sugar-cane, etc.) also include vertigo in their symptomatology. We cannot examine here the variety of *vertigo of pasture*, which seems to be also of a toxic nature.

Symptoms. The horse slackens its gait or stops suddenly; some subjects shake the head, straighten it or carry it sideways; they tremble, stagger, spread the legs, throw themselves aside, go backward, pull upon the tether, lean against the neighboring horse, the partition of the stall, the pole, or the harness if they

¹ Several times I have found in the ears of horses the larvæ of certain insects, probably those of the *Simulium cinereum*. Is it then the true cause of this condition? Without having still further proof, I am induced to believe it. Their frequency in the spring, when they are undergoing transformation, and the success which I have obtained in many cases by the injection into the ear of chloroform (1 to 5), justify in part this opinion. It is upon this point that research should be continued.—L. T.

are hitched. They are anxious, restless, and covered with sweat; they stumble, turn on themselves, sink upon the ground, and act in an uncontrollable way, or make unsuccessful efforts in order to take a standing position; soon, however, they rise again, manifest some symptoms of weakness for a few moments, and then return to a normal condition. During the attack the cerebral functions are troubled or cease, the mucous membranes and the skin are insensible, the pupils are dilated, the pulse and respiration are accelerated; sometimes we observe involuntary micturition and defecation. Vomiting is frequent in the dog.

The duration of vertiginous attacks is very variable (from one to two minutes on an average, about five minutes in serious cases); it is generally much shorter than in epilepsy.

The absence of spasmodic contractions in vertigo permits us to distinguish it from epilepsy. For a differential diagnosis, see Epilepsy.

Treatment. The treatment varies with the cause. This must be sought for and removed or alleviated as far as possible. Horses in which vertigo is occasioned by a prolonged stay in the stable may be cured by daily walks or work. The blinds should be unpolished and dull on their ocular side; it is often of advantage to change their fitting. Baker has recommended protecting the eyes from the light by means of a piece of leather of the size of six to twelve centimetres, stretched over the forehead from one blind to the other. The harness should be very exactly fitted to the parts which it supports. Horses that work in a circle should have their eyes covered.

When the attack occurs it will be advisable to step to the horse's head, cover the eyes and take off the harness. When falling becomes imminent we must, if possible, try to have it take place upon soft ground and clear this from all objects which might occasion wounds. When cold water is at hand we may, by head douches, shorten the duration of the attack or even arrest it.

As prophylactic means it is recommended to produce, from time to time, slight derivations upon the intestine by means of laxatives. Bleeding should be prescribed.

ADDENDA TO THE DISEASES OF THE NERVOUS SYSTEM.

Tic.

Nature. Tic (tic with support, tic upon the jaw) must be considered as a habit which consists essentially in the swallowing of air or of mouth foam which is formed by a mixture of saliva and air. As early as 1664 Solleysel considered tic as a vicious habit, which he compared to the practice of *smoking, chewing, or snuff-taking* in man. It is frequent in the horse, but rare in the ox; a few cases have been seen in the pig. Well-bred horses, thorough-breds, army horses, and studs are more specially affected, although draught horses are by no means free from it.

At the time air is swallowed respiration is interrupted; the larynx and base of the tongue are carried backward and downward by a contraction of the muscles of the larynx, tongue, and pharynx (omo-hyoideus, the sterno-thyroideus, and sterno-hyoideus; the pharynx becomes filled with air; then, when the larynx and base of the tongue return to their normal position, the air is ejected outward or is swallowed; at this time we hear one or two *bruits of the tic*, which are similar to those of *eructation*. The ancients thought that the abnormal brnit of cribbing was always due to a true eructation as it is produced in the ox; but this opinion is now rejected.

Etiology. The etiology of cribbing is complex, and its causes are of a variable nature:

1. This habit may be developed from the outset, under the influence of idleness. When the horses are lonesome in the stable they try to amuse themselves by licking the manger, partition walls, etc., by biting objects which are within their reach, by playing with the chains or weights of their tethers, etc.; these actions are especially observed if, during the day, the animals are closely tied. Other bad habits may be acquired in the same way. Abnormal motions of the mouth, suction of air and saliva, frequently precede the appearance of cribbing. When the horses work with activity and regularity they have no time for cribbing, and they rest as soon as they are in the stable. In a regiment of cavalry, where a large number of horses were addicted to cribbing, Pansecchi (1866) saw

the habit disappear almost entirely at the time of manœuvres (one single animal continued to crib); but as soon as they reached their quarters the horses were again affected by their mania.

2. Besides the spontaneous origin, irritation certainly plays a prominent part; in this way the habit may be communicated to entire stables.

3. Hereditary transmission of cribbing has been recognized. Collin observed it in forty-five descendants of an Anglo-Norman horse: one colt was affected at the age of three months, another at seven months, a third at eight months, five from the tenth to the twelfth month, and the largest number during the second year, some only after the third year.

The affection has been attributed to the excitement produced by a too violent action of the currycomb or by a very tight surcingle; at the time when these causes are acting we often see the animals bite their manger with all their strength and hold it tight between the incisors.

Among the causes mentioned we find also over-feeding and drinking water which is given in too small quantity (training of race and hunting horses); the latter circumstance compels the animals to lick continually the manger, walls, etc. (Sing). Hertwig admitted that tic was always preceded by a localized feeling of disturbance of the digestive organs or a slight indigestion; but this hypothesis does not seem to be well founded; the chronic alterations of the stomach which are frequently found at the autopsies of horses affected by cribbing (dilatation, chronic catarrh, thickening of the gastric walls, etc.) are rather consequences than causes of the habit.

Symptoms. I. IN THE HORSE we distinguish two principal varieties of cribbing: *cribbing which is performed while resting the jaw upon the manger*, and air cribbing.

Subjects which are affected by the first form support the incisors of one or both jaws upon the edges or bottom of the manger, more rarely the chin or the laryngeal region (Kunow); but the manger does not represent the only point of support; certain subjects lean upon the partition, the tether or halter chain, leather belts, carriage-pole, front quarters, the knee, and even the foot. Some animals seize different objects between the teeth, such as the edge of the manger, nails, etc. Günther has seen colts cribbing upon one of their mother's cannon. We have observed a horse which held with

all the teeth, and as far as the commissure of the lips, the edge of a stone manger. At the time the animal is about to swallow air it pushes against the object upon which it rests, opens the mouth and bends the neck; the muscles of the anterior edge of the neck become spasmodically contracted and prominent under the skin; we may hear one or two bruits of eructation; then the animal abandons its hold, and the act is over.

The horse in which *cribbing is skyward* does not hold any point of support with its teeth. He moves away from the manger, brings the head closer to the chest, executes with the lips a few particular movements similar to those which are performed at the time of prehension of food, then he lifts his neck suddenly, extends the head and swallows the air, producing a more or less loud bruit.

In the beginning the symptoms generally consist in a certain play of the lips and tongue; then we see a more and more marked tendency to lick neighboring objects or to suck saliva. The frequency of cribbing is extremely variable. Its manifestations may be separated by intervals of one hour, one minute, or only four or five seconds. There are animals that crib in the stable or when they are not watched; others are affected by the habit as soon as they are resting; others also do it immediately before or after eating, or again during the meal; in this latter case they throw the food that has been distributed to them all around. The subjects in which cribbing is most alarming are those which swallow air till they are much inflated by it. Ordinarily the habit disappears at once when the animals are sick or when they are placed in exceptional conditions.

The principal inconveniences caused by cribbing are: wasting of food, disturbing of neighboring animals, and the possibility of its propagation by imitation. Permanent symptoms (gastric or intestinal), properly speaking, are wanting in the majority of cases. Sometimes, however, cribbing produces chronic troubles of digestion and nutrition, gastric or intestinal tympanites and colics; this is the cause of the admitted close relations between this habit and affections of the digestive apparatus.

In old cribbers, Hell and Hartenstein have constantly found a hypertrophy of the sterno-maxillary and omo-hyoideus muscles (physiological hypertrophy).

II. In the ox we also observe different forms of cribbing. The most common is expressed in the following manner: the animal

raises its head and opens its mouth ; the tongue performs constant movements ; a frothy saliva is first accumulated on the commissures of the lips and soils the inner fascia of the jaws, then it is swallowed at the same time as *air bols*, which produces a gurgling noise. When this act has been repeated a certain number of times the rumen is distended by ingested air ; then cribbing ceases, and the gases are gradually ejected by eructation.

Weinmann has observed a cribbing bull which remained immobile, with its mouth closed, and which sucked air by the commissures of the lips, producing a particular bruit ; once meteorized, cribbing ceased ; then the air was ejected by eructation.

Johns has related the history of a cow which leaned its head upon a corner of the manger, and contracted the muscles of the tongue by sucking air and producing a particular and repeated gurgling bruit. As soon as meteorization had occurred, she remained entirely immobile during thirty to forty seconds ; then she extended her head upon her neck and appeared to feel a certain pleasant sensation in ejecting the air in a continuous stream.

III. In the clinic of Dorpat we have observed several cases of cribbing in the PIG. The animals put their incisors upon the edge of the trough and produced a bruit which was similar to that accompanying the act in the horse.

Diagnosis. The diagnosis of cribbing has a certain importance from a legal standpoint. This habit is, in fact, admitted to be serious enough to set aside a contract of sale in several countries (Bavaria, Würtemberg, Baden, Hesse, with a delay of eight days). The two principal diagnostic symptoms are a contraction of the inferior cervical muscles and the particular bruit which accompanies it. When the animals are accustomed to the examiner, he may recognize the gaseous wave in the œsophagus by applying the ear and hand upon different points of the course of the œsophagus. The wearing of the teeth, with its special characters, is sometimes observed in animals which do not swallow any air. It is especially important not to confound this habit with several cribbings which are performed without any support or with certain motions of the lips which have become a habit.

Treatment. Prophylaxis requires isolation of the animals affected by this habit. A radical cure is only possible in the beginning ; inveterate cribbers are incurable. Numerous means of treatment have been tried. The principal are :

1. Punishment: Of doubtful effect.
2. Laborious work, avoiding long rests: Favorable results.
3. Removal of all objects which may serve as points of support, fixation of the head upon a rack or in a very low position, etc.: Effects which are of short duration.
4. A manger, bag, or pouch placed upon the ground: Doubtful effects.
5. A mobile manger provided with a lead lining; also steel points upon the edge and bottom of the manger: Doubtful effects.
6. Use of a narrow muzzle: Favorable results for a certain time.
7. Anti-cribbing collars: It is generally sufficient to strap tightly the laryngeal region with a leather strap. This procedure has the inconvenience of compressing too much the vessels of the region. Ringheim and Burdajewitz have advised the use of special straps, which are wide and provided with a small metallic tongue, a steel spring, and sharp nails; but these latter may produce phlegmonous wounds and tumefactions of the region.
8. Günther's anti-cribbing bit: It is a hollow metallic cylinder which is provided with lateral openings. Several anti-cribbing halters have also been recommended.
9. Section of omo-hyoidean (Gerlach) or sterno-maxillary (Hertwig, Hell) muscles. The researches of Hering, Bassi, etc., have shown that the effects of this myotomy are not permanent.

Balking.

Nature. This habit, as found in the horse, is characterized by an obstinate refusal to execute an order which is neither abnormal nor unusual. It renders the horse dangerous and depreciates its value considerably. A distinction has been made between *absolute* and *relative* balking. The first renders the animals unfit for any kind of work. In the second, the animals are only stubborn to one kind of service. We recognize the following varieties: balking under saddle or in harness, balking single or double, etc.

Balking is particularly common in mares, also in Polish, chestnut, and sorrel horses; these latter have the worst reputation concerning this habit.

Etiology.—Balking is generally due to the brutality or ignorance of the trainer and to excessive correction; nervous, well-bred horses are very sensitive in this respect.

Constant irritation of the mouth, produced by a brutal hand, and painful wounds of the skin may determine it. As an occasional cause, we may mention horsing in the mare. The influence of heredity and pathologic conditions of the brain are but imperfectly known.

Symptoms. The symptoms of balking are sometimes active, at other times passive. There are cases where these abnormal phenomena coexist.

In *active balking* the animal stops during work and obstinately refuses to go any further. He stamps upon the ground, throws himself aside, sometimes upon his mate; he kicks, tears the harness, and breaks the pole or damages the front of the carriage; often the reins become caught between the hind legs. Some animals throw themselves upon the ground and move their four legs violently. The saddle horse tries to dismount its rider by backing or rearing, and may overthrow itself. Great excitement is always observed; the eye is threatening, the heart tumultuous, the mucous membranes of the head red, the superficial veins greatly distended. We remark trembling, abundant exudations, and an acceleration of respiration; in some rare cases balking is changed into veritable mania, and becomes marked by serious cerebral troubles. This phase of excitement is succeeded by a considerable depression of strength. In the stable the animals generally seem gentle and quiet.

In *passive balking* the animals also stop during work; they are as if nailed to the ground, and absolutely refuse to advance, no matter whether gentle or violent means are used; most of the time they try to turn back. If left to themselves, they start again after some time; in other instances they have to be unhitched and allowed to return to the stable.

These two forms of balking present a number of varieties. Very often the animal submits when it is not loaded heavily, or when going toward the stable, and it appears most stubborn when a hill must be ascended or when it is heavily loaded.

Diagnosis. This habit being admitted as sufficient to set aside a contract of sale in certain countries (Prussia, delay 4 days; Saxony, 5; Hesse, 9; Austria, 3), it is important not to confound it with any other affection. We must insist upon this point, that balking consists of a *conscious* refusal to perform a certain work answering to the conditions of conformation and training of the

animal. We may have to differentiate balking from immobility; this affection is characterized by *unconsciousness* of any abnormal movements.

We must not consider as a *balker* the horse which refuses to obey when undergoing exaggerated, unaccustomed exertions, or when a region of the body upon which the harness is supported is sore (wounds made by the collar, saddle, etc.).

It is important not to confound balking with the habit which is expressed by the word *shyer*. A medium-sized horse may pull upon a good road from 1500 to 2000 kilos, upon a bad road 1000 kilos, and upon a paved road 2500 kilos.

Treatment. Inveterate balking is incurable; but in benign cases, and when the habit is of recent date, we may, by kindness and patience, obtain a certain amelioration. Violent methods are always hurtful.

Nervous Diseases of Birds.

Among these diseases the most important are common cerebral hyperemia, or hyperemia which is complicated by hemorrhage and epilepsy.

1. *Hyperemia and hemorrhage of the brain*, or apoplexy, are quite commonly observed in male birds at the time of mating, especially when mechanical influences act upon the brain. They may also affect young poultry which are exposed to the sun's rays. Friedberger has also recognized these during the course of diphtheria. Their symptoms are as follows: vertigo, staggering gait, stupefaction, dilatation of the pupil, circling or backing movement, agitation of the wings or legs, particular epileptiform spasms, which may often be stopped by a simple touch. In some cases the head and neck are kept thrown back; the birds fall; the legs and wings are subject to convulsions which impart a rolling movement to the body; later the birds will stand up, at other times the trouble has an apoplectiform termination. As treatment, we give two tablespoonfuls of castor oil, or 0.1 gramme of calomel, or 0.5 gramme of jalap root (Zürn).

2. *True epilepsy* appears to be very rare in birds. Hertwig has mentioned a case of reflex epilepsy produced by intestinal worms. Friedberger observed attacks of true epilepsy in a chaffinch.¹

¹ Hartmann, Gay, Trasbot, and Mégnin have mentioned cases of epilepsy in birds.
—M. D. T.

The bird used to fall upon the bottom of the cage, and was subject to convulsive movements of the wings and legs; it would lie either on the back or abdomen, with its legs spread; the head was held up or thrown backward; the bill and eyelids were opened and closed alternately; the ocular globe turned around in the orbit. By degrees these phenomena became less intense and the bird would regain its equilibrium. Its condition seemed to improve by the administration of bromide of potassium (1 per cent. solution given in drinking-water).

[NOTE ON THE TREATMENT OF TETANUS.]

From a limited experience in the use of gelsemium in tetanus, the results have been more than encouraging; in fact, it appears to exert a decided and immediate improvement in these cases; in every instance brought to my attention up to the present time, where gelsemium has been used in the treatment of tetanus, cure has been obtained. There is no rule for the administration of the drug in this disease; it must be pushed as rapid as possible up to the *poison line*, as it is only its toxic action which exerts a beneficial influence upon the tetanic spasm; this fact was clearly demonstrated only yesterday (January 3, 1895); the case was a yearling with tetanus from castration; he was found with a marked spasm of the glottis and was struggling painfully for breath—in fact, was almost at the point of suffocation; hypodermatic administration of about 0.08 gramme of gelseminine (Merck's) gave positive relief in about half an hour. A second dose was then given, resulting in absolute relief within an hour; the animal was then left almost entirely free from spasm. He had been receiving half ounce doses of the fluid extract of gelsemium every half-hour for the previous twenty-four hours.

The danger of fatal poisoning is very slight, as muscular relaxation and disappearance of the spasm will be noticed before a sufficient quantity of the drug has been taken to produce this result. These facts are related with the hope that they will induce practitioners to use this drug in their tetanus cases, *pushing it to the poison line* and recording their results, as more light is needed on this question.—W. L. Z.]

SECTION III.

DISEASES OF THE RESPIRATORY APPARATUS.

I. DISEASES OF THE NASAL CAVITIES.

ACUTE CORYZA.

Acute Nasal Catarrh of the Horse.

Etiology. Acute nasal catarrh may be determined by various causes.

1. *Primitive* or *idiopathic* nasal catarrh is usually produced by cold. Young, weak, sickly animals are predisposed to it. It is much more frequent in the horse than in other domestic animals, the ox especially; this is mainly due to the numerous causes of taking cold to which the horse is exposed, and to the large size of its nostrils, which exposes the mucous membrane to external irritating agents. Among these latter we must particularly mention street dust and dusty food, smoke, fungi and spores which are flying in the air when the food is distributed; lastly, the ammoniacal atmosphere of badly-kept stables. The epizootic character of the disease points to the intervention of infectious elements, but we do not possess any positive facts to that effect.

2. *Secondary* acute nasal catarrh occurs quite frequently as a phenomenon of other affections of the respiratory apparatus or of infectious diseases. In some cases it is the result of an extension to the pituitary membrane by continuity of tissues of a neighboring phlegmasia (pharyngitis).

Symptoms. Acute nasal catarrh is indicated at the start by a diffuse petechiæ or hyperemia and a dryness of the mucous membrane; besides the expired air is a little warmer than usual on account of the congested state of the mucous membrane (inflammatory stage of irritation). Within a few days the principal symptom appears—a *discharge*. The mucous membrane is shiny,

tumefied, and œdematous (excretory stage). The nasal bilateral discharge is at first serous, clear, and of a watery consistency; it preserves its characters in benign cases, but when the catarrhal process is lengthened it becomes mucus-like and turbid, on account of very abundant epithelial desquamation; when white corpuscles are added to it in large numbers it assumes a purulent aspect (serous, mucous, and purulent catarrh). The animal sneezes frequently; the edges of the nostrils are covered with crusts formed by drying of the muco-purulent discharge. The inter-maxillary ganglions are sometimes tumefied (this is probably a symptom of the infectious nature of the catarrh).

The average duration of acute coryza is from eight to fifteen days. Ordinarily recovery takes place without the appearance of any secondary trouble; the discharge diminishes gradually, and later disappears.

There are cases where the disease is announced from the onset by alarming symptoms: chills, inappetence, a slight rise of temperature, acceleration of the pulse, and sometimes the process invades certain neighboring regions (sinus, eyes—by extension along the lachrymal canal); we then observe complications which are more or less serious—catarrhal conjunctivitis and photophobia, laryngitis, stomatitis, catarrh of maxillary sinus, etc. Acute nasal catarrh may become chronic.

Treatment. The mild form does not require any intervention; it ends rapidly in a cure.

In severe cases fumigation with hot water, to which is added a small quantity of cresol or phenic acid, constitutes an excellent local treatment. Proper ventilation of the stable and exercise in the fresh air when the weather is favorable have a marked influence upon the course of the disease. Green fodder, turnips, etc., or putting out on pasture, is recommended. Astringent liquids injected into the nasal cavities and the administration of anti-catarrhal remedies (salts, chloride of sodium added to powdered aromatic plants) are additional means of treatment.

CHRONIC CORYZA (CHRONIC NASAL CATARRH OF THE HORSE.

Etiology. As an essential disease it is much more rare than acute catarrh; it is generally developed at the expense of the latter;

when the intensity of the phlegmasia and the tendency to invade are much accentuated, or when its producing cause acts for some time. *Secondary* chronic nasal catarrh is much more frequent and more important. The morbid processes which determine it are :

1. Glanders. This disease frequently develops with the clinical appearances of chronic nasal catarrh. In case this latter is detected in a horse, it should be watched, and all necessary precautions taken in order to avoid all danger of contagion.

2. Catarrhs of the diverticula of the nasal cavities (maxillary and frontal sinus, and the guttural pouches), diseases which are marked by an obstinate discharge.

3. Tumors, parasites, abscesses, etc., of the nasal cavities.

4. Diseases of teeth (suppurating alveolar-dental periostitis and its complications).

5. Chronic diseases of the respiratory apparatus in general, and chronic constitutional diseases (leukemia, anemia, etc.).

Symptoms. In chronic nasal catarrh the pituitary membrane is pale or cyanosed; it becomes thickened and presents varicose tracts when the disease is of long standing. The discharge may be gelatinous and mucous, "similar to frogs' eggs," or purulent; it becomes agglutinated to the wings of the nose; its color varies from a dirty to a yellowish-gray; it has frequently a fetid odor; its quantity is very variable; at times the pathological secretion gives place to a true blennorrhœa, at other times the discharge is only abundant at intermissions (when working or when the head is kept low); often it ends by furrowing upon the floor of the nasal cavities a small tract where the integumentary pigment is wanting. In a general way, a unilateral discharge indicates that the catarrh is secondary, symptomatic (glanders, neoformations which are localized upon one nasal cavity only, dental diseases, etc.); when the catarrh is idiopathic the discharge is usually bilateral. In the first case the lymphatic intermaxillary ganglions are almost always tumefied and indurated.

Ulcerous erosions constitute a complication which is wholly peculiar to chronic nasal catarrh; they are only observed in cases where the trouble is very old; these are losses of substance of the mucous membrane the dimensions of a pin's head or of a lentil, all are superficial and defined by sharp edges that are not thickened, characters which differentiate them distinctly from glandered ulcers. But we must observe that they are sometimes seen in chronic glan-

ders complicated by secondary nasal catarrh. Their existence, therefore, does not permit us to exclude glanders. Ulcerous erosions (formerly designated under the name of catarrhal ulcerations) heal without leaving any cicatrix, which enables us also to distinguish them from glandered ulcers.

Chronic coryza may persist for weeks, months, or even years. It is generally difficult to cure. When its duration is prolonged it is sometimes complicated by hyperplastic growths of the nasal mucous membrane.

Diagnosis. It is especially important to know if nasal catarrh is idiopathic or symptomatic, and this question is far from being always easy to solve. The course of the disease may furnish some information; in the primary form it is much more regular than in the secondary. We must especially take into account the *unilateral* or *bilateral* character of the discharge. There are cases where the nasal mirror permits us to make important observations, but we are sometimes obliged to trephine the sinus or even inoculate the suspicious catarrhal secretions. (See Glanders; Differential Diagnosis.)

Treatment. It is essentially local, and comprises the following means: fumigations with hot water, cresol, phenic acid, creosote, tar, essence of turpentine (they may be tried in turn in the order mentioned); astringent injections in the nasal cavities (sulphate of zinc, cresol, phenic acid in a 1-2 per cent. solution; a $\frac{1}{2}$ per cent. solution of the nitrate of silver, etc.). These injections are made either with a syringe, or by means of an atomizer, and by the nostrils or by an opening made with the trephine (Dieckerhoff).

The usefulness of medicinal agents which are administered internally (ammonia, essence of turpentine) is at least doubtful. The animals must be given good food, and be placed in well-ventilated stables, or be set at liberty in the open air; the manger should be kept perfectly clean, the nostrils have to be cleaned frequently, and the skin must be kept with care: such are the principal hygienic indications to be observed.

Horses affected with chronic nasal catarrh must be isolated. All direct interference with the ulcerous erosions must be avoided; it would have an effect upon the evolution of these lesions, and make them take on other than their typical characters; it would, therefore, render more difficult a diagnosis of chronic nasal catarrh and of glanders.

[Peroxide of hydrogen sprayed into the nasal cavities with an atomizer will give excellent results in this disease. This method of application brings it into contact with every part of the mucous membrane of the nose and sinuses; this should be followed by insufflation into these cavities of iodoform, with which may be associated small quantities of sulphate of zinc, acetate of lead, nitrate of silver, etc. The sneezing which this will produce will cause the powder to be carried into the most distant parts of these cavities; cure is usually obtained in from four to six weeks.—W. L. Z.]

NASAL CATARRH OF THE SHEEP.

Malignant Catarrhal Fever; Glanders of the Sheep.

Under the denomination of nasal catarrh several very different affections are described in the sheep, which authors designate by the expressions *sheep glanders*, *benign* and *malignant catarrhal fevers*, which were then considered as nearly synonymous terms. Malignant catarrhal fever has, indeed, all the characters of an infectious disease, of which the affection of the pituitary is only a phenomenon. We will examine successively: 1. *Acute nasal catarrh*. 2. *Chronic nasal catarrh*. 3. *Malignant catarrhal fever*.¹

1. *Common acute nasal catarrh* of the sheep is an insignificant disease; classical treatises do not mention it. It is produced by cold (spring, fall), by inhalation of dust, etc. Its symptoms are: a nasal discharge, sneezing, and a wheezing respiration.

2. *Common chronic nasal catarrh* has been described by most authors under the name of "benign catarrhal fever." It follows acute catarrh when the causes which have produced the latter are permanent (damp weather, cold winds, etc.). In young and weak animals it may in course of time endanger life. It is quite common in some herds, and is characterized by a permanent mucopurulent discharge which agglutinates the wings of the nose and forms crusts.

3. *Malignant catarrhal fever* of authors, "sheep glanders," is evidently an infectious disease. It is spoken of here in order to

¹ There has also been observed in the rabbit a malignant catarrhal contagious fever which is often fatal, and mainly characterized by a rhinitis which is due to coccidias which are found in innumerable quantities in the mucous membranes of the nose and pharynx, in the tympanic cavity, and in their products of secretion (Zürn).—N. D. T.

point out more the difference which exists between this affection and the acute and chronic forms of common nasal catarrh.

NATURE OF MALIGNANT CATARRHAL FEVER OF THE SHEEP. Malignant catarrhal fever of ovines, which is an infectious, contagious disease, presents a great analogy to variola in the dog. It is characterized by alterations of the different mucous membranes, principally of the conjunctival, pituitary, and tracheo-bronchial mucous membrane.

Etiology. According to the researches of Friedberger, the agent of this disease is an infectious principle which may be perpetuated for years in the same stable, and which may be transported to some distance. We have seen the disease break out in animals of sheep-folds which were entirely separated from those in which it existed. It is generally observed in an enzootic or epizootic state; it is feared with good reason. Formerly it was believed that common coryza could *degenerate* and become *malignant*, or be transformed into *glanders*. Some authors have described an epizootic form of benign catarrhal fever.

Symptoms. In the beginning we recognize the symptoms of purulent nasal catarrh; the neighborhood of the nostrils is coated with a thick mucus; the wings of the nose are frequently agglutinated; when they become compressed, a muco-purulent discharge at times runs out of it, in other cases the discharge is fetid; the nasal mucous membrane is red and tumefied. To these phenomena are sometimes added manifestations of laryngitis (cough), bronchitis, and, in serious cases, those of broncho-pneumonia or traumatic pneumonia.

In the eyes we may observe blepharitis or purulent conjunctivitis, keratitis, ulcerations and abscesses of the cornea. The eyelids are red, tumefied, and agglutinated by a purulent, viscous matter; the hairs upon the inner angle of the eye are moist and agglutinated by a brownish-yellow viscous substance. The conjunctivæ are reddish-yellow or cinnabar color; sometimes they secrete in abundance a purulent exudate (blennorrhæa). In the neighborhood of the sheath we observe a purulent dermatitis and ulcerations; there exists at the same time a purulent preputial catarrh. In the digestive apparatus we observe the symptoms of a catarrhal phlegmasia; inappetence, constipation or diarrhea, and expulsive efforts.

The general condition is greatly disturbed; the fever is slight, but the animals are depressed and emaciated; they rapidly become

weak and anemic; their gait is staggering; at certain times they fall and remain stretched upon the ground in a condition of deep sleep. Sometimes this comatose state is interrupted by clonic contractions of the extremities and muscles of the trunk, grinding of the teeth, etc.; these are all symptoms which announce a fatal termination. The course is generally acute; the prognosis is very serious, especially in lambs.

Pathological anatomy. The upper regions of the pituitary are furrowed by reddish streaks or show a diffuse red tint; the glandular part is scarlet-red, black, or blue, and shiny upon its surface, also tumefied and covered by a gelatinous, dirty-yellow, creamy, granular, or caseous exudate; in the other territories of the respiratory apparatus we find lesions of catarrhal bronchitis, inflammatory œdema, traumatic pneumonia or broncho-pneumonia. The liver is clay-colored, doughy, friable, and infiltrated with fat; the renal epithelium is affected with fatty degeneration; the brain is œdematous; the heart is soft, friable, reddish-gray; the muscles are pale; there are ecchymoses in the various organs. The blood is liquefied or slightly coagulated. The cadaver is much emaciated.

Treatment. The principal prophylactic measures consist in isolating the patients, giving them nutritious food (crushed corn and oats), and in disinfecting the sheepfold. In benign cases we may administer tonics (sulphate of iron) and bitters in an electuary. In serious cases there is no use trying to cure; killing the patients when done early is the most economical plan.

NASAL CATARRH OF THE OX, PIG, AND DOG.

Nasal catarrh is rare and of slight gravity in the ox and pig; but it is very common in the dog. Secondary chronic nasal catarrh is sometimes observed in animals as an epiphenomenon of several infectious or invasive diseases (variola of the dog, tenoid pentastoma, strongylus, malignant catarrhal fever, etc.). We do not possess any precise information upon the idiopathic nasal catarrh of the ox or pig. In the dog this disease is marked by a discharge, sneezing, wheezing, and rattling bruits at the time of inspiration or expiration; sometimes the discharge is hemorrhagic or purulent. Its principal cause seems to be the action of cold. The distinction between primary and secondary catarrh (variola) is not always easily made.

No treatment is needed in most cases. Inhalations of salt solutions (chloride of sodium), ammonia, cresol water ($\frac{1}{2}$ to 1 per cent.), and the internal administration of saline dissolvents (chloride of sodium, sulphate of sodium, ammonia, etc.), favor a cure.

SERIOUS INFLAMMATORY CONDITIONS OF THE NASAL MUCOUS MEMBRANE.

Phlyctenular, Croupous, Follicular, Diphtheric Inflammations.

These serious inflammations are produced by intense irritations, most of which are of a specific nature. They are almost always the expression of an infectious disease.

The anatomical classification leaves much to be desired where *croupous*, *diphtheric*, *phlyctenular*, and *follicular* inflammations are concerned. These phlegmasia, in fact, do not at all represent clinical entities, and they may be determined by different causes. Thus, while croupous rhinitis is produced either by external mechanical or thermic irritations, or by the infectious agent of strangles, this latter disease may be accompanied by phlyctenular, croupous, or follicular rhinitis. Nevertheless, from a practical standpoint, it is proper to preserve the anatomical division, for at the present time the infectious agents which generate these various lesions are unknown.

I. PHLYCTENULAR INFLAMMATION OF THE NASAL MUCOUS MEMBRANE occurs mostly as a phenomenon of strangles (phlyctenular strangles) or of acute nasal catarrh. Phlyctenular rhinitis is a "vesicular catarrh," that is to say, a disease in which, under the influence of the maceration to which the elements of the Schneiderian mucous membrane are subjected, or to rapid transudation, there are produced upon the surface of the mucous membrane small vesicles which may pass into a pustular condition. When they burst and their contents dry up there are formed in their place thin crusts, which are cast off as soon as cicatrization of the tissues is completed.

II. CROUPOUS INFLAMMATION is generally a consequence of a violent irritation of this membrane [inhalation of smoke and hot air during conflagrations (Trinchera), etc.]. In a few rare cases it is observed during the course of strangles. Nasal croup, which exists

at times in an enzootic state in new army horses, appears to be due to a specific pathogenic agent; this form of croupous rhinitis must therefore be considered as an infectious disease. Röhl and Bruckmüller, who have had occasion to observe it quite frequently in Vienna, have given a good description of it. The nasal mucous membrane is covered with a croupous membranous exudate, which is reddish-yellow and sometimes of large extent, and is gradually eliminated through suppuration; it is much hyperemied, and when the croupous membranes have become detached it is found to be excoriated and bloody. We recognize besides a yellowish viscous nasal discharge, an obstruction of the respiration, fever, inflammatory tumefaction of the lymphatic vessels of the upper lip, cheeks, and lower jaw, as well as of the submaxillary ganglions. The termination is generally favorable. The treatment is confounded with that of common nasal catarrh; when the disease takes on an infectious character, it is advisable to isolate the patients.

III. FOLLICULAR INFLAMMATION is an intense phlegmasia of the mucous glands of the pituitary and of the sebaceous glands of the skin in the neighborhood of the nostrils. The excretory canals of these glands are obstructed, and the cellular and fibrinous inflammatory products become accumulated in their interior. At this period we remark upon the surface of the mucous membrane small pimples which slowly ulcerate. We do not refer here to a lymphatic folliculitis which may be compared to that determined by tumefaction and suppuration of the agminated follicles of the intestine. Lymphatic follicles exist, in fact, neither in the nasal mucous membrane nor in the cutaneous integument; the expression "folliculitis" would then be improper in order to denominate this process. The affection seems to be differentiated from croupous rhinitis by its localization only; very often, though, both diseases exist at the same time. Follicular inflammation of the pituitary is evidently the result of a specific cause. Among the cases observed in practice there are some the manifestations of which appear identical with certain lesions of strangles, and the others seem to represent an infectious disease which is similar to this latter affection. It is usually observed in an enzootic state in military training stables and in localities where a large number of horses are congregated. We have found it several times in livery stables. Its contagiousness is beyond doubt. In army horses we have seen it communicated directly from sick to healthy animals.

The symptoms begin with a violent, acute, nasal catarrh, which determines in the mucous membrane of the nasal partition alterations which are characteristic. The surface of this membrane is rugous, and covered with small yellowish pimples, which are formed by the tumefied mucous glands; at the same time that these pimples ulcerate they become covered and surrounded by an adherent membranous exudate; after elimination of this we find in the place of the pimples numerous superficial ulcerations, with an intense red border, which are healed by an epithelial proliferation starting from their periphery; the pimples heal rapidly without leaving any trace. We may recognize similar pimples and ulcerations upon the edges of the nostrils and wings of the nose, on the skin of the upper lip, upon the inferior border of the masseter, etc.

From these ulcerations lymphatic cords originate which extend to the ganglions of the submaxillary space, which are always greatly tumefied, or they are prolonged as far as the lymphatic ganglions of the neck and presternal region. We have observed a lymphangitic cord of the thickness of an arm, which was extended all along the mastoido-humeral as far as the entrance of the chest. These tumefactions of the large lymphatic vessels of the neck, like those of the head, are remarkable for their tension and their excessive hardness; they form abscesses quite frequently. Often, also, we have recognized conjunctivitis, which was accompanied by blennorrhœa.

The disease is generally benign. In most cases the cure is complete within two to four weeks. Confusion with glanders is easily and surely avoided by a serious examination of the patients. The existence of numerous superficial ulcerations which heal rapidly and without leaving any cicatrix, their frequent extension to the cutaneous integument, concomitant lymphangitis, the mildness of the process, etc., permit us to make a diagnosis with perfect certainty. By simple examination of the buccal cavity we may differentiate this disease from *pustulo-contagious stomatitis*, which is invariably accompanied by typical lesions of the buccal mucous membrane.

The treatment is that of croupous rhinitis (fumigations of hot cresol or phenicated water, etc.). Upon the ganglions and tumefied lymphatic vessels we must apply iodoform, cresol, or phenicated ointment. As a rule, we use the following mixture: Iodo-

form or cresol, 2 grammes; paraffine ointment, 20 grammes. Some authors recommend touching the ulcers with nitrate of silver. We do not advise resorting to this practice, as it has the inconvenience of rendering more difficult the differential diagnosis of rhinitis and glanders.

IV. DIPHTHERIC INFLAMMATION OF THE NASAL MUCOUS MEMBRANE has nothing in common with diphtheria of man. It must be considered as a necrotic inflammation of the pituitary. As a primitive affection this disease is very rare, and it is only developed under the influence of irritation of a violent character in order to determine a mortification of the mucous membrane. It is almost always symptomatic, and appears as a phenomenon of several infectious morbid conditions (acute glanders, petechial fever of the horse, malignant catarrhal fever of the ox, etc.).

Dickerhoff observed in the horse four cases of very distinct diphtheric inflammation of nasal and buccal mucous membranes and of the external integument of the lips and nose. He saw at the same time all the symptoms of a general infection of the organism, an intense fever, gastric symptoms, a reddish-yellow coloration of the mucous membranes, hemoglobinnria and albuminuria, as well as erysipelatous tumefactions of the legs and chest. The disease terminated in death within three to six days.

TUMORS OF THE NASAL CAVITIES.

Independently of glandular lesions we may find in these cavities, upon the median septum or ethmoidal sinuses, tumors and abscesses which are accompanied by chronic nasal catarrh. These lesions are generally unilateral. Often the neoplasms become ulcerated and determine a tumefaction of the lymphatic submaxillary ganglions. In the horse they are of great clinical interest on account of their possible confusion with glanders.

I. FIBROMATA. These appear sometimes in the shape of polypi which are pediculated or sessile, at other times as wide and flattened neoformations. Polypous fibromata are sometimes visible externally; they maintain a chronic nasal catarrh and determine a wheezing or rattling respiration, which is due to a contraction of the corresponding nasal cavity, which they obstruct entirely at times. In some cases their superficial layer suppurates and becomes loose; there is also a bad-looking discharge; the submaxillary ganglions

of the corresponding side are tumefied. When these tumors continue to develop they may penetrate into the buccal or guttural cavities and encroach on the nasal wall.

Fibromata in the form of blotches render the mucous membrane very irregular and lead also to chronic catarrh; the contraction of the nasal cavities is a hindrance to respiration; they become separated by an ulcerating process and give place to a discharge of bad aspect. In a microscopic examination of the altered pituitary membrane Grawitz found an amyloid degeneration of the mucous glands, of the connective-tissue fibres, and of the vascular walls.

II. SARCOMA AND CARCINOMA. These are more rarely observed than fibromata. They displace the bones (maxillary, sub-nasal, palatine) and may advance into the buccal or guttural cavities, where they interfere with mastication and deglutition; some become ulcerated and produce a fetid ichorous discharge; the sub-maxillary ganglions are tumefied. The sarcomata, as a rule, start from the periosteum of the bones which constitute the walls of the nasal cavities (osteosarcoma). Carcinomata frequently occasion epistaxis, and may be accompanied by metastatic alterations of the lymphatic ganglions of the tongue.

III. ANGIOMA. Angioma of the nasal wall is produced by a double mechanism, by a dilatation of the normal vessels and by a vascular neoformation (simple angioma); they frequently acquire a cavernous character (cavernous angioma). It is never distinctly defined, but diffuse and much extended in surface. Its coloration varies from brown to bluish-red. It easily produces hemorrhages under the influence of mechanical irritations or of an augmentation of the blood-pressure (violent exertions), and it has a remarkable tendency to ulceration. It is often found covered with blood-clots.

When the process retrocedes and the neoplasm disappears it always leaves a cicatrix similar to the chancre of glanders. Ulceration of angioma is accompanied by a bloody discharge of bad aspect and by a tumefaction of the intermaxillary ganglions.

From this description we may draw the inference that angioma of the nose may be easily confounded with glanders; in some cases the diagnosis could not be established otherwise than by a microscopic examination of the tumor.

IV. HYPERTROPHY OF THE SINUSES. It is congenital or due to chronic nasal catarrh, to strangles, or chronic osteitis starting from

the dental alveoli. This hypertrophy may hinder respiration considerably ; it is always accompanied by chronic nasal catarrh.

V. ABSCESES. These are found in the nasal walls and ethmoidal sinuses. They are generally of traumatic origin ; sometimes, however, they are developed during the course of catarrhal inflammation of the pituitary ; they occasion a destruction of the cartilages and bones, and are characterized by a purulent discharge, by epistaxis, by tumefaction of the ganglions of the tongue, etc.

VI. AS RARER NEOFORMATIONS, we must also mention lipomata, osteomata, adenomata, actinomycoma, tubercular neoplasms, amyloids, etc. ; dermoid cysts, tumors which are produced by the *Strongylus armatus* and, in the dog, hairy growths which are developed upon the pituitary.

EPISTAXIS.

Nasal Hemorrhage.

This is a symptom common to several diseases. Epistaxis or nasal hemorrhage is observed in all domestic animals, but particularly in the horse.

Etiology. The causes of epistaxis are numerous :

1. Traumatism, injuries of mucous membrane, contusions, etc. ;
2. An abnormal blood-pressure after violent exertions, forced races, or in cerebral congestions ; blood stagnation in the veins of the nose during the course of cardiac diseases, pulmonary diseases, etc. ;
3. Diseases of the bloodvessels ; varicose dilatation of veins, and especially angioma of the nasal mucous membrane ;
4. Neoformations (see preceding chapter) and ulcerations upon the pituitary ;
5. Serous inflammations of this mucous membrane ;
6. General diseases which are accompanied by hemorrhages : strangles, hemophilia, petechial fever, anthrax, leucocythemia, etc. In the horse slight epistaxis, which does not occur frequently, is generally a symptom of glanders.

Quite frequently the cause of epistaxis cannot be recognized during life.

Symptoms. As a rule, nasal hemorrhage is unilateral. At times the blood exudes in drops or runs in a thin thread ; at other times the discharge is simply marked by reddish streaks. The

blood is never frothy, and the cough is wanting. In the majority of cases epistaxis is passive and periodical; it may exceptionally lead to death.

Treatment. Slight hemorrhages do not require any treatment. In serious hemorrhages we may first try styptic solutions (perchloride of iron in a solution of 5 to 10 per cent.). When epistaxis becomes dangerous by its abundance, we must tampon the nasal cavity with cresol, phenicated or sublimate wadding. In order to do this successfully it is sometimes necessary to resort to trephining. Internally, we may administer styptics: salts of lead, tannin, sulphate of iron, ergot, etc.

II. DISEASES OF THE ACCESSORY CAVITIES OF THE NOSE.

INFLAMMATION OF THE MAXILLARY AND FRONTAL SINUS IN THE HORSE.

From the standpoint of the frequency of the inflammations which are developed in the mucous membrane of the sinus, they must be classified in the following order: superior maxillary (antrum of Highmore), inferior maxillary, frontal, ethmoidal, and sphenoidal sinuses. The disease is generally unilateral.

Etiology. Catarrh of the maxillary and frontal sinus is usually chronic. It may be produced by traumatisms (fractures, contusions, etc.), or colds, but it is mostly the result either of the propagation to the mucous membrane of the sinus of an inflammatory process which starts from the nasal cavities or from the dental alveoli, or by disorders produced in these cavities by neoformations developed in it. In some cases it is due to glanders or rickets (colt).

Pathological anatomy. In the beginning the mucous membrane of the sinus is tumefied, injected, congested, or of a bluish color; between the mucous membrane and the bone we find a yellowish, gelatinous exudate; the bones and cutaneous integument are hyperemic. If the disease is somewhat prolonged, the mucous membrane becomes thickened and secretes a serous, mucous, and, later, a purulent liquid. The opening which joins the sinus with the nasal cavities (median meatus) becomes tumefied and obstructed; the exudate accumulates in the sinus, becomes thickened and decomposed; sometimes pus collects between the inflamed mucous

membrane and the bony wall upon which it rests; this becomes polished, thinned, swollen; and the cavity of the sinus becomes enlarged. The partition which exists between the maxillary sinuses becomes thinned, and even disappears entirely. In exceptional cases the purulent exudate works its way out, forming a fistulous tract. The pus may also burrow toward the alveoli and produce dental caries. (This communication is sometimes established by a propagation to the sinus of a phlegmasia which is at first localized to the dental arcades; then it is not rare to find the sinus filled with alimentary matters). The dilatation of the frontal sinus may compress the brain and produce a partial atrophy of this organ. The same modifications are produced upon the ethmoidal cells and upon the sphenoidal sinus.

Symptoms. The characteristic symptoms of catarrh of the maxillary and frontal sinus are:

1. *A unilateral discharge*; it is the characteristic manifestation. In the beginning and when the inflammation is very intense this discharge is sometimes bloody. Its quantity and consistency are variable; at first muco-purulent, thick and odorless, later it becomes mucous, granular, and fetid. It flows abundantly during exercise and when the head is kept low.

2. *Unilateral tumefaction of the ganglions of the tongue*, which gradually become indurated on account of the proliferation of the peri-vascular connective tissue of the cavernous system.

3. *Dilatation of the maxillary and frontal sinuses*. According to Haubner, this dilatation is preceded by tumefaction and hyperemia of the skin. We easily recognize swelling of the bones of the face by examining comparatively the diseased and the healthy side. In one horse Köhne found the external wall of the maxillary sinus swollen, prominent, fluctuating, and forming a prominence of the size of half a cocoanut.

4. *Dulness on percussion*, which is recognized upon the diseased side. It is due to an accumulation in the sinus of the product of catarrhal secretion and to a thickening of the mucous membrane. This symptom may be wanting when the exudate is yet slight. The negative result given by percussion would not help to exclude the existence of the collection in the sinus.

This disease has ordinarily a chronic course. It is often prolonged for months, even years. Among the accidents which complicate it we may mention cerebral symptoms, which occur

when the ichorous inflammation extends to the meninges and the brain itself (Friedberger), or if there is a purulent inflammation of the ethmoidal cells and sphenoidal sinuses (Lustig). In cases where these complications occur the animals show symptoms of immobility or of encephalitis.

Diagnosis. Besides these clinical characters the practitioner, in order to make his diagnosis, may resort to the trephine; this operation is recommended by Haubner in cases of suspected glanders. In common catarrh the mucous membrane of the sinus is smooth or slightly roughened; it is irregular or rugous when chancres have developed in it; besides, in this latter case the glandered process invades almost constantly the detached cutaneous strip upon the orifice which has been opened by the trephine. We must, however, add that this operation does not give a positive result in all cases. (See Glanders; Differential Diagnosis.)

Treatment. Success is only attained by practising trephining. Inhalations, which are beneficial in chronic nasal catarrh, are without effect in the collection of the sinus. After the opening of these cavities the pathological liquids run off as they are secreted, and we may apply directly to the mucous membrane such therapeutic agents as are indicated. The operation assures a cure, except in cases of serious alterations of the mucous membrane or bones, tumors, necroses, etc.

It is not necessary to trephine both maxillary sinuses, but only the upper; we may also apply the trephine to the partition which separates this sinus, so as to render both accessible by one and the same opening.

Into the cavities we make injections of solutions of cresol, sublimate, phenic acid, sulphate of zinc, nitrate of silver, tar, etc.; care should be taken to tampon the edges of the opening in order to keep them apart. The injections must be made upon the standing animal, as in those which are recumbent some portion of the inflammatory exudate may be drawn into the lungs, and thereby determine a broncho-pneumonia.

[In the early stages of this disease it will frequently yield to the treatment of nasal catarrh; indeed, some practitioners of experience make the statement that insufflation of the nasal cavities with iodoform is sufficient to cure the worst cases of collection of the sinuses. My own experience is that local application of iodoform and nitrate of silver will cure many cases, especially when the dulness of the

accumulation in the sinuses, cannot be demonstrated by percussion (iodoform, 5 parts; magnesium carbonate (light), 5 parts; nitrate of silver, 0.5 to 1 part). When there is dulness on percussion the trephine should always be used, opening the lower portion of the inferior maxillary sinus, which insures the most perfect drainage. This one opening is sufficient for all purposes of treatment, which in all uncomplicated cases will result in cure. A discharge of pus from the trephined sinus is not always immediately manifest, even when there is marked dulness on percussion; this fact should not lead the practitioner to think that he has made an error in diagnosis.—W. L. Z.]

CHRONIC CATARRH OF THE GUTTURAL POUCHES IN THE HORSE.¹

Œstres Disease of the Sheep.

ŒSTRES VERTIGO : FALSE TURNSICKNESS.

Œstres disease is due to the presence of larvæ of the *Œstrus ovis* in the maxillary and frontal sinuses and inside the bony core which serves as a basis to the horn. It is marked by symptoms of chronic catarrh of the nose and sinus and by certain cerebral troubles.

NATURAL HISTORY. The œstre of the sheep is a yellowish-gray diptera which is almost bare; it is about one centimetre in length. It is very common in Germany, and is especially frequent in dry and hot summers; it exists in sheepfolds, in the neighborhood of localities where the sheep are on pasture, on the borders of woods and in bushes. The œstres swarm during the warmest hours of the day from July to September. The fecundated females seek the flocks and fall upon them. The animals become suddenly anxious and run off, holding the head between the forelegs, or they place themselves in a circle and crowd against one another with the head lowered. The insects deposit their progeny in the neighborhood of the nostrils. The affected sheep move about and rub the nose against their legs, the ground, or hard objects. These phenomena of excitement disappear rapidly and the animals present the appearance of perfect health for nearly nine months.

The larvæ which are deposited upon the edges of the nostrils reach the nasal cavities, the frontal and maxillary sinus, and the

¹ See Vol. I., p. 42.

cavities of the horns. It is there that they reach their maturity toward the ninth month. They are very small and capillary in the beginning, having the length of two to three centimetres at the time of maturity; they are then constituted of eleven rings, which are of a brownish-yellow color and marked with blackish transverse striæ upon their upper fascia. The cephalic ring is fitted with two buccal hooks, between which is found the buccal dimple. The larvæ, on reaching maturity, emigrate from March to May (rarely before or after); this migration occasions certain disturbances, which are due to irritation of the mucous membrane of the nose and sinus; we observe more rarely phenomena of cerebral excitement (disease of œstres larvæ). Twenty-four hours after its exit from the sinus the larva becomes a caterpillar; six to seven weeks later the insect takes its flight.

Pathological anatomy. We find in the sinus a variable number of larvæ (10 to 100) during the various periods of development; they are surrounded with mucus and blood. The mucous membrane is tumefied, red, infiltrated with blood, covered with purulent and bloody mucus; sometimes it is gangrenous; on the points where the larvæ are fixed it exhibits rounded depressions, which are surrounded by a cushion. The meninges are hyperemic and the brain is œdematous. The parasites may traverse the ethmoidal plate and become located in the brain; they stray at times as far as the pharynx, larynx, or trachea.

Symptoms. The symptoms appear in the springtime, a period during which the larva reach their maturity. In the beginning we observe a serous discharge, which later becomes mucous and is sometimes streaked with blood; the animals sneeze frequently and may expel the parasites which are undergoing their migration.

The head is constantly shaken; the patients scratch the nose with the front legs, or they rub it against hard objects which may be within their reach. We frequently find, as a consequence, a skinned and bleeding forehead; sometimes the whole face is the seat of a swelling which renders it deformed. We recognize, besides a catarrhal inflammation of the conjunctiva, tumefaction of the eyelids, and watery eyes.

In benign cases we do not observe any other phenomena; in serious cases we remark cerebral depression. The patients are affected by vertiginous attacks (œstres vertigo); at times their gait is regular; sometimes, but much more rarely, they walk sideways, describing

circles (false turnsickness). The existence of this latter symptom has been doubted. (See Observation of Gilis.)

When the disease is allowed to pursue its course it ordinarily ends in death. This is preceded by epileptiform spasms, grinding of teeth, etc., which occur mostly through asphyxia. The patients succumb, as a rule, from the fourth to the eighth day.

Differential diagnosis. Œstres disease may be confounded with turnsickness, whence the name "false turnsickness" which has been given to it. The symptoms of nasal catarrh—sneezing, discharge, blows with the head, swinging movements, and catarrhal conjunctivitis—complete the diagnosis.

Treatment. Prophylaxis consists in keeping the animals in the sheepfold at the time of the swarming of the œstres. But this measure is impracticable in the majority of cases. We may try to ward off the insects by smearing the nose of the animals with tar, fetid animal oil, cresol, etc., immediately before driving them to pasture. According to Zürn, the beneficial influence of these agents is very doubtful. The larvæ and nymphs must be destroyed.

In order to hinder the ascending progress of the larvæ into the nasal cavities authors have advised for a long time tobacco snuff, powdered hellebore, violet and carline roots, which are blown into the nose by means of a feather quill. This procedure is only efficient when applied soon after the larvæ have been deposited upon the edges of the nostrils. If once they reach the sinus, the parasites can only be removed from it by trephining. Zürn advises resorting to this operation in valuable animals only, for the extraction of all the parasites is seldom attained; they offer perfect resistance to the action of medicinal agents. (The operating field is located upon both upper angles, which are formed by an intercrossing of the median line of the head and of a transverse line which passes through the centre of the orbits. We may also amputate the horns.) But in all cases killing is the most advisable course.

LINGUATULA TÆNIOIDES IN THE NASAL CAVITIES AND SINUS OF THE DOG.

NATURAL HISTORY. The *Linguatula tænioïdes* or rhinaria (*Pentastoma tænioïdes*, Rud.) is a vermiform parasite of the order

of Arachnides, which was discovered in 1757 by Chabert. The history of its development was distinctly detailed in 1856 by Leuckart. This author has demonstrated by experiments that the *Linguatula denticulata*, which was formerly considered as a separate species, is only the larval form of the *Linguatula tænioïdes*. This latter is located in the respiratory tracts of carnivora, especially in the dog, and its denticulated larva is developed in the viscera of herbivorous animals.

1. *Linguatula tænioïdes* is most frequently met with in the dog and wolf, in the nasal and frontal sinus (maxillary sinuses are wanting), and by preference in the cul-de-sac of the median meatus (Colin); it is found besides in the ethmoidal cells, pharynx, larynx (below the vocal cords), and in the middle ear (Gellé). In the other animals (horse, mule, sheep, goat) and in man it is extremely rare. Its length varies from 2 to 13 centimetres (female, 8 to 13 centimetres; male, 2 to 3 centimetres). Its body, which is of lanceolate form, is often rolled up, and is wider in front than in the back; it is white or yellowish-white in color; it is divided into about ninety rings, which give a tæniform aspect to it. The head, which is spheroidal, shows at its lower part a rounded opening, which is surrounded by a horned ring: this is the mouth, around which are four linear openings destined to contain the four biarticulated members, and which are provided with a kind of terminal claw. (These slits were formerly taken for mouths, from which the name of pentastome was given to the parasite.)

2. The *Linguatula denticulata* is a larval form of the preceding; it is often found upon the sheep, but it exists also in the hare, rabbit, ox, stag, camel, etc., and in man. It is found encysted in the liver, the mesenteric ganglions, kidneys, and lungs, and free in the abdominal cavity and respiratory tracts. It is elongated, flattened, whitish, transparent, and about half a centimetre long, and is formed of eighty to ninety segments, which are all provided with fine hooks (whence it derives the name "denticulata"); the legs, which number four, are provided with chitinous pieces on the double claws; the genital organs are rudimentary.

The evolution of the *Linguatula tænioïdes* is well known at the present day. The adult linguatulæ, which are located in the nasal cavity of the dog, copulate; each female produces about a half a million of eggs; these, which are ejected upon grass, fodder, leaves, etc., with the nasal mucus, may be ingested by herbivorous

animals. In the stomach of these latter the shell of the egg is dissolved; the embryos, the form of which reminds one of the acarus, are about one millimetre long; they are provided with from four to six legs and a tail. At a given time they perforate the intestinal wall and become located in the parenchymatous organs: liver, mesenteric ganglions, kidneys, etc. Once encysted, the parasites undergo several metamorphoses; in about six months they reach an ultimate larval state (*Linguatula denticulata*). Soon they perforate their cystic envelope and migrate toward the abdominal cavity, whence they have a tendency to escape to the outside by reaching the lungs and bronchial tubes; in some cases they determine thus a fatal traumatic pulmonary œdema (Gerlach). When their host is killed before this period the larvæ may be ingested by a meat-eating animal. According to Gerlach, their resistance to the different destructive agents is considerable; desiccation does not diminish their vitality. They reach the nasal cavities of carnivorous animals by the nostrils or pharynx; after an abode of four or five months in these cavities they reach their maturity. The *Linguatula denticulata* has been transformed into a *Linguatula tenioïdes*.

Pathological anatomy. The mucous membrane of the nasal cavities and sinus shows various alterations; tumefied, red, inflamed, and gangrenous, it is covered by a muco-purulent or bloody exudate.

In some countries the linguatulæ seem to be extremely rare. They are much more common in Berlin than in Munich. Hering observed them but once. Out of 630 dogs which were examined at Alfort, by Colin, 64 were affected by these parasites; their number varied from 1 to 11.

Symptoms. The presence of linguatulæ is indicated by manifestations of chronic nasal catarrh. The animals are affected with a discharge and sneezing; they rub the nose against hard bodies or scratch it with their paws; they do not seek for the food which may be scattered over the ground, and take only that which is given to them. In some subjects we observe a slight bleeding from the nose which is almost continuous; eggs or even linguatulæ may be ejected with the mucus. Breathing is rattling; we notice at certain times dyspnœic or asphyxic attacks. Sometimes the sense of smell seems to be entirely abolished. Emaciation may occur rapidly. In some patients the linguatulæ perforate the palatine and produce a very abundant salivation (Adam, Perdan).

In benign cases they are ejected one after the other, and the disease ends in cure. In others the preceding symptoms are complicated by serious cerebral disturbances which are sometimes impossible to distinguish from rabid phenomena. In some instances the animals are anxious and over-excited; they bite, howl, scatter their litter, pass the muzzle through the bars of their cage or turn upon themselves; at other times they are apathetic and stupefied from the start; occasionally the lower jaw is paralyzed. In this serious form death may result in a few days. At the autopsy we recognize hyperemia, œdema, and a slight inflammation of the cerebral substance.

The presence of eggs of linguatula in the nasal discharge is an important diagnostic symptom.

Differential diagnosis. The disease which is produced by linguatula must be differentiated from hydrophobia, from common catarrh, and from canine distemper. The diagnosis is based upon the recognition of parasites either *intra vitam* or *post mortem*.

In all cases where we proceed to the autopsy of animals which presented rabiform symptoms, it is advisable to examine the nasal cavities carefully. Some observations (Friedberger, Perdan) seem to demonstrate that linguatula may produce serious general symptoms, and even death, without the existence of important alterations upon the mucous membrane of the nasal cavities or upon that of the sinus.

Treatment. Trephining of the sinus and nasal cavities may alone produce a radical cure. The opening which is made upon the sinus is also useful when the linguatulæ are located in the nasal cavities; parasitocides (cresol, benzol, phenic acid, etc.) injected into the sinus run partially through the nostrils. The efficacy of fumigations of tar vapors and of different snuffs is most uncertain.

Linguatula denticulata occasions, as a rule, no alteration in the animals in which it lodges. However, Leuckart in his experiments has recognized (in the hare) pulmonary and hepatic lesions which were caused by this parasite. The invaded organs were inflamed, studded with hemorrhages, and pierced by numerous intricate galleries.

III. DISEASES OF THE LARYNX.

LARYNGITIS.

Inflammation of the mucous membrane of the larynx may be observed isolated or coexisting with a phlegmasia of the neighboring organs (mucous membrane of the pharynx, sinus, and trachea). The expression "laryngitis" should only be used to denominate it in cases where the laryngeal symptoms dominate the scene; in all other circumstances the term "catarrh or inflammation of the anterior respiratory tracts" is preferable.

Laryngitis assumes *catarrhal*, *phlegmonous*, *croupous*, *diphtheric*, and *ulcerous* forms. From an etiological point of view it is *essential* or *symptomatic*, *traumatic*, *tuberculous*, or *glanderous*.

If considered according to its course, it is *acute* or *chronic*.

We shall limit ourselves to describing the following three forms:

1. Acute laryngitis.
2. Chronic laryngitis.
3. Croupous laryngitis.

1. ACUTE CATARRHAL LARYNGITIS: ACUTE CATARRH OF THE LARYNX.

Acute catarrh of the laryngeal mucous membrane, which is generally designated under the name of angina, is particularly common in the horse and dog—animals which are more exposed to various external irritations than all others.

Etiology. In primitive acute laryngitis we generally recognize cold as its cause (breathing cold air, drinking cold water, sudden chilling by rainstorms, draughts, etc.). It is especially seen in the spring and fall; it often has the type of an epizootic disease. As predisposing conditions we may mention debility and want of exercise. Among the other conditions which are apt to produce it we must also mention influences of a traumatic or chemical order which are exerted upon the laryngeal mucous membrane (compression of the larynx, continual barking, drugs, smoke, acrid chlorine or sulphur vapors); *cough occasioned by swill*, and observed in animals to which this residue is given for the first time, appears to be of an inflammatory nature; certain substances which are contained in this food, especially alcohol, seem to produce

upon the laryngeal mucous membrane an irritation to which this membrane becomes gradually tolerant.

Secondary acute laryngitis occurs as an epiphenomenon of several general diseases (strangles, disease of young age), of contagious pneumonia of the horse, and serious affections of the head, etc. It is also frequently the result of the extension to the larynx of a phlegmasia which is at first located in the nasal or pharyngeal cavities, trachea, or bronchial tubes.

Pathological anatomy. The principal anatomical alterations are tumefaction, redness, and ecchymoses of the laryngeal mucous membrane, which is covered with a serous, mucous, or purulent exudate. According to Bruckmüller, the upper region of this membrane is particularly affected; therein are found a purulent destruction of the mucous glands and superficial ulcers upon the epiglottis and vocal cords.

Symptoms. The principal symptom is a *dry*, harsh, painful cough, which comes by spells in the beginning, and which later becomes moist and mucous. It is produced by cold air at the time the patients are taken out, by the ingestion of liquids and fodder, and also when the subjects rise suddenly or when they are much excited (this is often observed in hospitals when the owners call to see their animals).. In the dog the mucus loosened by the cough is immediately swallowed.

Acute catarrhal laryngitis is also indicated by an abnormal sensitiveness of the laryngeal region; it may easily be detected by pressure, which the patients seek to avoid, as it produces coughing spells. As a consequence of the tumefaction of the mucous membrane and an œdematous infiltration of the dilators of the glottis, respiration becomes laborious; we sometimes hear at a distance a contracting râle or wheezing bruit; auscultation of the larynx permits us to detect a moist râle. To these symptoms are added later those of pharyngitis, bronchitis, or rhinitis: inappetence and dysphagia, dryness of the buccal mucous membrane, a nasal discharge, and in some cases a slight fever; the former invariably indicates a complication. Intense hyperemia must always awaken suspicion of a serious infectious disease.

There has been described¹ an *enzootic laryngo-tracheal catarrh* of the horse, which existed in a very intense form in the German army

¹ Preuss. Militärrapporte, 1888; Zorn: Adam's Wochenschr., 1888; Rust: Cölner Naturforscher versammlung, 1888.

during the summer of 1888, and also affected a great many private horses. (In Prussia it was observed in twenty regiments belonging to ten different army corps.)

1. **PATHOLOGY.** This disease was distinguished by its extreme contagiousness; often all the horses of the same stable or of the same division were affected within a few days.

It existed particularly in animals which were in a convalescent or cured condition from contagious pneumonia or influenza. Its period of incubation lasted a few days. Infection took place through the medium of expired air. We have seen animals contract the disease after having been led within a few steps of the diseased subjects.

2. **SYMPTOMS.** The principal and constant symptom was a dry, violent acute cough (more rarely weak and painful). The trachea and larynx were very sensitive to palpation; this latter immediately produced a violent coughing spell. Soon a slight nasal discharge appeared, which was serous in the beginning, later mucous; sometimes we remarked also a slight tumefaction of the lymphatic ganglions of the submaxillary space. Auscultation and percussion of the thorax have not generally revealed anything abnormal.

In most of the affected horses we noticed a more or less intense febrile reaction, which was announced from the beginning of the disease by chills lasting for several hours; the temperature rarely reached 41° C. Out of 426 sick horses which were observed by Rust, this veterinarian found 173 times a temperature exceeding 39° C. Animals in which the fever was intense had a variable appetite and an accelerated respiration (20 to 24 per minute), the sensitiveness was dulled and weakness quite pronounced.

3. **COURSE.** In the large majority of cases the disease was benign; its duration varied from eight to fourteen days, and the affected subjects were able to begin work after a week of convalescence; this has rarely been longer. Exceptionally, when the animals were obliged to make violent exertions or when exposed to cold, several complications were observed: colics, intestinal catarrh, pneumonia, and pleurisy. In several cases this affection coexisted with contagious pneumonia.

4. **TREATMENT.** As treatment hygienic means only were resorted to: rest and walking in open air. Some veterinarians (Zörn) have advised favoring or producing infection of all the horses which

are exposed to contagion (in regiments) in order to shorten the duration of the epidemic.

This laryngitis is evidently a particular infectious disease which has not yet been described.

Differential diagnosis. Cough is a symptom common not only to the several forms of laryngitis, but also to most other diseases of the respiratory apparatus (tracheitis, bronchitis, pneumonia, pleurisy). No matter what its characters may be, we are not able to make a diagnosis of laryngitis; we must especially take into account the sensitiveness of the larynx to pressure and the laryngeal respiratory bruits. Tracheitis and bronchitis are indicated by tracheal or bronchial râles. Pneumonia and pleurisy are accompanied by phenomena which are of importance in the physical examination of the thorax.

It is not always easy to determine the degree of gravity of the laryngeal inflammatory process. As a general rule, we must base our judgment upon the intensity or mildness of the local symptoms and fever, upon the general condition, the nature of the exudates, and abnormal respiratory noises.

Laryngoscopy, which nowadays has reached a high degree of perfection in human medicine, is only used in a restricted way in veterinary practice. In poultry and cats and dogs with short muzzles we may examine the larynx and the upper region of the trachea by opening the mouth wide, depressing the tongue by means of a spatula, and lifting the laryngeal region. In dogs with long muzzles the operation is difficult; it is still more so in large animals. These latter must be placed in a recumbent position, and the larynx illuminated by means of reflected light. In proceeding thus we can explore the glottis. But in subjects of large species laryngoscopy is quite laborious and too complicated to become common in our practice.

Treatment. The principal prophylactic indication consists in proceeding gradually in the training of young subjects. The patients must be allowed to rest in well-ventilated stables, with a mild and equable temperature. Externally we use, as a rule, moist heat. Priessnitz's compresses must be applied upon the laryngeal region. This treatment often gives good results. Hot water fumigations are also of advantage. Internal medication is not curative; we must be satisfied in combating the symptoms. For the dog we prescribe :

Hydrochlorate of morphine	0.1-0.2 gramme.
Bitter almond water }	20 grammes.
Distilled water }	

Three doses of 10-15 drops daily.

We may dissolve these agents in 300 grammes of distilled water, and give every day three tablespoonfuls or teaspoonfuls of the preparation.

[Iodide of potassium alone or in combination is a valuable agent in the treatment of laryngitis. Spirits of turpentine may also be used to advantage, especially in dogs, given in emulsion in from five to ten drop doses. The best results will probably be obtained by the use of cocaine and codeia given in solution in bitter-almond water. The following formula will be found most satisfactory :

Cocaine hydrochlorate	2.00 grammes.
Codeia	5.00 "
Aqua amygdalæ amaræ	360.00 "
Dose for the horse	15 to 30.00 "
Dose for the dog	10 to 20 drops.

Given every four or six hours the most distressing symptom of the disease (constant cough) is almost instantly relieved.—W. L. Z.]

2. CHRONIC CATARRHAL LARYNGITIS.

Etiology. Chronic catarrhal laryngitis is usually observed in the dog and horse—especially in the spring and fall. It is commonly designated under the names of *convulsive cough*, *irritating cough*. Of the canine species, old animals, long-haired poodles, lap dogs, king Charles, and retrievers are predisposed to it.

In a majority of cases chronic catarrhal laryngitis succeeds the acute form. The *action of cold* being its principal determining cause, explains the frequency of the disease and the epizootic character that it sometimes presents in the spring and fall. After certain traumatic irritations and a few acute infectious diseases (distemper), a chronic inflammatory state of the laryngeal mucous membrane may persist.

Tumors of the larynx: papillomata, polypi, sarcomata, carcinomata, actinomycomas, glanderous and tubercular neoformations, etc., often maintain a chronic catarrhal phlegmasia in the surrounding mucous territory. In one dog Nocard observed obstinate cough similar to that of chronic laryngeal catarrh, which was due to a compression of the vagus nerve by tumors of the mediastinum.

A few authors, Röhl among others, think that the convulsive or

irritating cough of the dog or horse is identical with whooping-cough (pertussis) of man. We do not agree with this theory, which, moreover, is not based upon any positive fact. Whooping-cough is an infectious, contagious disease which is considered by some as a mycosis of the tracheo-laryngeal mucous membrane, by others as a neurosis of the superior laryngeal. It is particularly frequent in children, is marked by coughing spells separated by intervals of variable duration, and it never returns.

The characters of convulsive cough in animals are quite different. It is not at all contagious, and principally affects old animals; it often returns and presents nothing typical in its course, nor has it ever been seen to coexist with epidemics of whooping-cough. Its epizootic appearance in the spring and fall is common to several catarrhal diseases; these latter may, indeed, assume an enzootic character under the influence of identical causes. Such are the reasons which have made us decide to associate the convulsive or irritating cough of animals with chronic laryngitis; besides, the autopsy shows almost constantly the alterations of the latter affection.

Pathological anatomy. The anatomical alterations of chronic catarrh of the larynx are similar to those of catarrhal diseases of the other mucous membranes. The laryngeal mucous membrane is thickened and rough; its vessels are dilated. At certain points we find papular blotches (proliferation of the papillary body); at others there are small pimples which give to the membrane a rugous aspect (hypertrophy of mucous glands, granular laryngitis); finally, in some places we observe small whitish circumscribed spots which are produced by an epithelial desquamation. The connective tissue is hypertrophied and overrun with white corpuscles. In man, in old chronic catarrh, the mucous membrane becomes thin and atrophied and its glands disappear (Ziegler).

Symptoms. The principal and often the only manifestation is *cough*. It is generally dry, coarse, croaking, more rarely moist and accompanied by a bruit of rattling contraction or similar to rhonchus. It is especially frequent and spasmodic during the night, which is undoubtedly due to the abundance of mucus deposited upon its surface. Ordinarily we find a certain degree of dyspnoea. The spells are sometimes followed by attacks of suffocation, retching, and even true vomiting; these phenomena are produced by an accumulation in the back part of the mouth of thick and viscid

mucus coming from the larynx. The head is stretched upon the neck and shoulders and carried low. The general condition is not disturbed. The animals are lively, the appetite is normal, there is no important febrile reaction. Auscultation and percussion of the lungs do not reveal any alteration of this organ.

Chronic laryngeal catarrh is of long duration; it may last for months, even years, and, in many cases, acute attacks of the trouble recur which alter the prognosis considerably.

Treatment. Coughing spells increase the hyperemia and inflammation of the laryngeal mucous membrane. The patients must be given absolute rest, and they should be protected from all causes of excitement. Application upon the larynx of hydropathic compresses (Priessnitz's compresses) has also been advised. Formerly derivative frictions (antimonial ointment, etc.) to the integument of the laryngeal region were recommended. These produce only a temporary effect and may become dangerous; it is better to reject them. The principal treatment consists of a direct action upon the laryngeal mucous membrane. But this local intervention will never give in domestic animals the results obtained in human medicine, on account of the technical difficulties that its application presents in veterinary medicine. We may resort to vapor inhalations (water, aromatic flowers, cresol, phenic acid, tar, essence of turpentine, 1 per cent. solution of chloride of sodium or ammonium, bromide of potassium, tannin, nitrate of silver), or to insufflation of powder (morphine; nitrate of silver 0.1 gramme, and sugar 1 gramme, etc.). It has also been advised to touch the mucous membrane with a brush or a small sponge impregnated with a one-fifth per cent. solution of nitrate of silver. In the horse for inhalations we use a sacciform mask which may be replaced by a blanket spread over the head. For the dog we may employ the same apparatus as for man; in veterinary schools large inhalation boxes are used.

Dieckerhoff, imitating the intra-tracheal injection method advised by Lévi, has quite recently recommended a direct injection of astringent liquids into the larynx. For the horse he uses a bent hollow needle with which he perforates the crico-tracheal ligament, with the point turned upward toward the glottis. The principal agents employed are alum in a solution of 0.5 per cent., and acetate of lead in a solution of 0.3 per cent. We can confirm the good effects which these injections sometimes give; but they are not

without danger.¹ In the dog we make the injection through the crico-thyroid ligament. For this animal we may complete the local treatment by the administration of expectorant remedies. We generally prescribe morphine and hydrocyanic acid (see Acute Laryngitis). In the dog it is also advisable to resort to solvents.

The following is a useful formula :

Hydrochlorate of ammonium	50 grammes.
Chloride of sodium }	āā 100 “
Liquorice powder }	

To be given at each meal in a tablespoonful dose.

3. CROUPOUS LARYNGITIS.

Croupous laryngitis, which is also known under the denominations of *laryngeal angina*, *membranous angina*, and *croup*, generally spreads to the trachea as far as the origin of the bronchial tubes; very often also phlegmasia is propagated to the pharynx, as it is also transmitted to the larynx in cases of pharyngitis. Nevertheless, in this affection the larynx is the organ where the inflammatory phenomena are most pronounced.

AFFECTED ANIMALS. Croupous laryngitis, which is frequent in the ox, is rare in the horse and cat, and rarer still in the subjects of other species. Bruckmüller only observed it in the horse and ox. We have seen it several times in the cat. According to Spinola, it does not exist in the pig.

Etiology. In the majority of cases croupous inflammation of the laryngeal mucous membrane seems to be produced by cold. It has been attributed especially to sudden changes of temperature in mountainous regions, the remaining at pasture on cold nights, and the ingestion of frozen food or drink. The disease is very common in the mountains of Piedmont, Italy (Forneris). It is ascribed also to traumatic irritations of the mucous membrane, the action of foreign bodies, very hot air, smoke, hot drinks (ammonia is particularly dangerous in this respect); among the irritating gases are mentioned particularly chlorine and sulphurous acid. In a few instances the enzootic appearance of croupous laryngitis has made us suspect its infectious nature. It happens as a symptomatic

¹ Aruch advises dusting the larynx with powdered medicaments, which is done by means of a tracheal canula fitted to Richardson's atomizer. This author has often used this method without the least inconvenience, and he believes that it renders great service in the treatment of laryngeal diseases.—N. D. T.

morbid condition during the course of certain infectious diseases, such as "contagious headache of the ox," anasarca, acute glanders, etc. In fine, the laryngeal mucous membrane may be overrun by necrotic processes, accompanied by lesions of a diphtheric aspect.

The croupous character of the exudate is due either to the intensity of the causal influences or to the tendency to plastic exudations, so strikingly marked in the ox. (See Croupous Enteritis of the Ox.)

Pathological anatomy. In the beginning the alterations of the laryngo-tracheal mucous membrane consist in exudates which are disseminated and become confluent little by little, and end by forming a continuous pseudo-membrane which is grayish or yellowish-white; in the ox it is frequently brownish-yellow. These exudates, which sometimes seem as if moulded into the trachea and larynx, are easily detached. With the microscope they are found to be composed of fibrinous filaments, white corpuscles, and free nuclei. According to the cases the mucous membrane is hyperemic, studded with hemorrhages, tumefied, or infiltrated with pus. The subjacent connective tissue is œdematous.

As complications of croupous laryngitis we may cite: tracheitis, bronchitis and croupous pneumonia, pneumonia produced by foreign bodies, and pulmonary emphysema.

Symptoms. This disease is indicated by very serious symptoms, which appear suddenly. It ordinarily starts with intense fever and chills; the general condition is alarming; nostrils and mouth are opened; there is abundant salivation, coughing spells, and dyspnœa; the head is stretched upon the shoulder; the patients are anxious and keep their front legs apart and butt against the wall. The mucous membranes are red, the eyes prominent, and the cutaneous veins distended. The neighborhood of the larynx is tumefied, hot, and sensitive to pressure; in compressing the throat and trachea we may produce dyspnœic attacks. Respiration is accompanied by many varied sounding bruits from contraction; the respiration is suspirious, wheezing, rattling, rhonchus. The hand, if placed upon the throat, perceives a quivering bruit which is produced by a circulation of air in the laryngeal opening.

As accessory troubles we may mention inappetence, cessation of rumination, salivation, regurgitations, constipation, and exhaustion of the milk secretion.

When the symptoms become rapidly aggravated, death by

asphyxia may occur within a few days. In some instances the patients are carried off by a secondary pneumonia.

In benign cases pieces of croupous membranes or membranous cylinders are ejected from the nose and mouth as early as the third, fourth, or fifth day; recovery takes place very rapidly. The duration of the disease is about one week.

Prognosis. In the ox and horse the prognosis must be guarded. Asphyxia may be produced from one moment to another, and in some serious cases recovery sometimes takes place rapidly. Our personal observations establish that the prognosis is relatively more favorable in the cat.

Treatment. Croupous laryngitis has a rapid evolution. It is rare that the means employed in its treatment succeed in arresting its course. We are limited to making hot water or quicklime water inhalations, and in applying Priessnitz's compresses upon the throat, hot cataplasms which ought to be renewed frequently (Weber), mercurial ointment or astringent liquids (solution of the nitrate of silver). When dyspnoea is intense we should not hesitate to practise tracheotomy; this operation is too often not performed in time. In the dog and cat it is advisable to give an emetic.

CEDEMA OF THE GLOTTIS.

Cedema of the Laryngeal Mucous Membrane.

Cedema of the glottis consists essentially of an cedematous tumefaction of the submucous laryngeal connective tissue, which sometimes presents the characters of a serous or purulent inflammation, at other times those of simple passive cedema.

Etiology. Inflammatory cedema of the glottis is *primary* or *secondary*. As a primary disease it may occur during the course of a severe laryngitis, as a consequence of lesions of the mucous membrane produced by foreign bodies or by acrid drugs, after a cold (sudden changes of temperature), or a prolonged walk in a dusty atmosphere (Bugniet). Secondary cedema is related to inflammation of a neighboring mucous membrane; it is a symptomatic manifestation. It is observed in severe laryngitis, anthrax, glanders, anasarca, variola, pyemia, and pneumonia (Schütz).

Common passive laryngeal cedema appears at the time of a consecutive stagnation in diseases of the heart, lung, or kidney, or it

may be caused by compression of the jugular veins. In the horse we have seen it produced several times by strangulation.

Pathological anatomy. Œdema is generally limited to the upper part of the larynx, that is to say, on the base of the epiglottis, the aryteno-epiglottic ligaments, the arytenoids, and the lateral ventricular ligaments; the lower regions, the glottis, and vocal cords are rarely affected, because at these points the mucous membrane is united to the subjacent parts without the intervention of a connective-tissue layer. The œdematous mucous membrane forms a thick, gelatinous, quivering swelling, which is more or less prominent in the larynx, and closes up the glottis at the time of inspiration. When it is incised a serous, purulent, or gelatinous liquid escapes, according to the case (serous, purulent, gelatinous infiltration). Sometimes the œdema extends to the neighboring regions—pharynx, base of tongue, and nasal cavities (Schütz).

Symptoms. The symptoms of inflammatory œdema of the glottis ordinarily appear quite suddenly, and much resemble those of croup: there is very intense dyspnœa, particularly noticeable during inspiration, which may lead to asphyxia; cough, râles, and more or less marked wheezing, which are perceptible at some distance; also anxiety, sweats, and a congested condition of the mucous membranes; the eye is fixed and prominent. The patients perish within a few hours if neglected.

Passive œdema has a slower evolution; it produces marked symptoms, which may continue from a few days to several weeks.

Diagnosis. Confusion between œdema of the glottis and croupous laryngitis is frequent, on account of the similarity of the symptoms of the diseases; the diagnosis of the former, however, may be established by taking into account the subacute evolution of the process and the absence of false membranes, which in croupous laryngitis are ejected by coughing.

Treatment. We must apply energetic counter-irritation to the laryngeal region by means of mustard. When these means are inefficient we have to resort to tracheotomy, and perform this operation quickly. Bleeding, powerful derivatives, local applications and inhalations of astringent solutions, pieces of ice, may be successful in subacute cases; but these means are inefficient when the evolution of the disease is very rapid.

ROARING, CHRONIC WHEEZING (ATROPHY OF THE DILATORS OF THE GLOTTIS.)

Laryngeal Hemiplegia.

AFFECTED ANIMALS. Unilateral atrophy of the dilators of the glottis (posterior and lateral crico-arytenoidean, posterior part of the arytenoidean) is a consequence of paralysis of the recurrent laryngeal nerve. This disease, which is commonly known under the name of wheezing, especially affects young horses between the ages of three and six years. Günther studied it from an etiological point of view in 1823, and gave a good description of it in 1834. The researches of Vatel, Dupuy, and Youatt, which were published before Günther's work, were, however, commenced after the first experiments of this author.

Paralysis of the recurrent laryngeal is not a disease which is found in the horse exclusively; it has been observed in the ox (Vitz, Ollman) and dog (Esser, Friedberger, Fröhner).

Etiology. In the majority of cases wheezing is due to heredity. Well-bred English stallions almost invariably transmit this affection to their progeny; this is a well-known fact in our studs. A long and weak neck seems to be in all breeds a circumstance which favors the development of wheezing. It has been claimed that in the case of thoroughbred horses it may be produced by training: under this influence the heart and aorta acquire a considerable development; in the thoroughbred the connective adipose tissue being sparingly developed, the recurrent laryngeal is more exposed to the action of the arterial pulsations, and may thus become atrophied by compression (Ellenberger).

The question why paralysis exists almost always on the left side (according to Günther, in 100 cases of laryngeal hemiplegia, 96 are found on the left side) has often been raised. As a rule, we ascribe this to the superficial position of the left recurrent laryngeal. But the intra-pectoral relations of this nerve seem to be more important, as Günther first pointed out. This author thinks that in the diseases of the chest, which are so frequent in the horse (contagious pneumonia), the left inferior laryngeal nerve, which crosses the aorta, participates in the morbid process, hence its paralysis. Franck, in his lectures, pointed out the possibility of a stretching of the nerve by the aortic impulse, which is strongly developed in well-bred horses.

Martin has quite recently indorsed this opinion. According to him, in thoroughbred horses the left recurrent laryngeal is irritated by the aortic impulse, and stretched by a pushing backward of the heart and the great development in the length of the neck. These authors have also ascribed as a cause the compression of the nerve between the aorta and trachea (Vaerst). According to Sussdorf, the left recurrent nerve shows constantly a flattening which may be seen with the naked eye where it passes between the aorta and trachea.

Contagious pneumonia is a cause that is relatively frequent in wheezing, which appears to be produced here by a propagation of the pleural inflammation to the inferior laryngeal nerve (Günther, Mölle, Vaerst, etc.).

The compression of this nerve by tumors—lymphomata, cicatrices, abscesses of the lymphatic ganglions during the course of strangles, dilatations of the œsophagus—may determine its paralysis (Bassi, Hugh Fergusson, Aubry, Esser). Bassi has shown that accidental wounds of the recurrent laryngeal in the region of the neck lead to the same consequences. In a case which has been related by Glöckner, wheezing occurred after a thrombosis of the carotid.

Certain cases of serious phlegmonous pharyngitis and laryngitis are accompanied by paralysis of the muscles of the larynx and contraction of the glottis. These affections may be accompanied by a chronic inflammation of the submucous connective tissue and contraction of the inter-arytenoidean space. As causes of laryngeal paralysis we must also mention chronic lead-poisoning, feeding with a poor quality of peas, vetches, and lucern; it is possible that these latter plants act through the medium of the mould with which they are sometimes covered.

Pathological anatomy. The dilator muscles of the glottis (left side) are atrophied and affected with fatty degeneration; they appear of a pale color, which gives them the aspect of connective-tissue fibres (predominance of myolemma). Atrophy, which is ordinarily incomplete, is particularly marked upon the posterior crico-arytenoidean; in general it corresponds exactly to the muscular territory innervated by the recurrent laryngeal. The direct consequence of this lesion is sinking of the left arytenoid, which partially obstructs the opening of the larynx. In a few rare cases it exists on the right side; sometimes, when occurring after serious attacks of laryngitis, it is bilateral. The crico-thyroidean is often irregu-

larly developed ; it seems to have undergone a kind of compensatory hypertrophy, as if to supplement the action of the degenerated muscles. We also frequently find the recurrent laryngeal atrophied, its elements having undergone yellow fatty degeneration. In certain cases the lungs are emphysematous.

Symptoms. The principal symptom is a bruit of laryngeal contraction perceptible at the time of inspiration ; it is due to the lowering of the arytenoid and paralysis of the left vocal cord, also to the narrowing of the glottis and upper orifice of the larynx.

This contraction bruit (*stridor*) is heard sometimes during rest (in severe cases). As a rule, it appears only at the time of exercise. According to its characters and sound, which are very variable, we say the horse wheezes, blows (*Rohren*), rattles, snores, croaks, bellows, etc. The nostrils are greatly dilated. Sometimes this bruit is very loud and perceptible at some distance ; it is rarely heard on expiration. It is almost always heard at its maximum of intensity after violent exercise (gallop) ; it disappears of itself within a few minutes when the animals are allowed to rest ; in order to stop it immediately it is sufficient to compress the nostrils and to proportion the inspired air to the calibre of the larynx. Dyspnoea increases in proportion to the intensity of the abnormal bruit. We may observe suffocating spells and falling. Chronic wheezing is often accompanied by a peculiar cough, which is sometimes buzzing ; at other instances it becomes quivering, when the vocal cord vibrates during the inspiration following a violent expiration.

Palpation of the larynx enables us to recognize atrophy of the muscles ; the left arytenoid being more easily pushed backward and downward than its mate ; this procedure produces at times wheezing during rest. According to Bassi, in examining the larynx by the mouth, which is kept open by means of a speculum, it would be impossible to recognize the displacement of the arytenoid or the relaxation and deviation of the vocal cord.

The disease has generally a slow course ; as a rule, it progresses steadily to a fatal termination. Sometimes an acute wheezing is developed in the course of certain diseases of the respiratory apparatus (laryngitis, contagious pneumonia). We have seen above that this latter disease frequently leads to chronic wheezing. Laryngeal hemiplegia is an incurable disease. Nevertheless, cases of paresis and paralysis of recent origin and consecutive to contagious pneumonia or to serious laryngitis may be cured. The

same is true in the toxic forms, in which wheezing disappears on the withdrawal of poisonous food. The mode of utilization of animals has a considerable influence on the course of the disease.

Differential diagnosis. Chronic laryngeal wheezing may be confounded with contractions of the nasal and guttural cavities which are produced by tumors (polypi), natural deformities, fractures, or by the collection of guttural pouches, etc. In all these cases palpation of the larynx furnishes precise information. We may remark that some healthy horses produce bruits similar to wheezing, and there are grooms who can imitate this noise in a striking manner (Günther).

Treatment is exclusively surgical. Günther formerly practised with some success a resection of the paralyzed vocal cord and arytenoid by means of an incision made in the larynx. Stockfleth and Bassi have also performed this resection, following the indications given by Günther. Quite recently Möller¹ has treated with success, by total extirpation of the arytenoid cartilage, wheezing due to unilateral atrophy of the muscles of the larynx. Tracheotomy and compressive apparatus, which are applied upon the nostrils, are palliative means.

Long-continued arsenical treatment has been recommended; but its effects are most uncertain. In the beginning we may try injections of strychnine in the neighborhood of the larynx (0.05 gramme to 0.1 gramme of strychnine per dose and per day); we continue these injections for some time, with an interval every third day.

From a prophylactic standpoint, all horses affected with chronic wheezing should not be used for breeding purposes.

For information concerning the study of wheezing considered as a disease setting aside a contract of sale, see treatises on legal veterinary medicine.

Spasm of the larynx, which is a very rare disease in animals, consists in spasmodic occlusion of the glottis. It is symptomatic of an affection of the upper laryngeal nerve. Its causes are unknown. Gerlach has described *spasmodic respiration* as a special form of chronic wheezing; but the spasmodic poisons mentioned by this author belong to the group of paralyzants of the larynx.

¹ Möller: Das Kehlkopfpfeifen der Pferde und seine operative Behandlung. Stuttgart, 1888.

Quite recently Degive¹ has observed a case of laryngeal spasm in a horse which was followed by asphyxia. It is necessary to combat this disease by hypodermatic injections of morphine.²

IV. DISEASES OF THE TRACHEA AND BRONCHIAL TUBES.

ACUTE BRONCHIAL CATARRH.

Acute Catarrhal Bronchitis.

FORMS OF BRONCHITIS. According to the intensity, duration, and localization of the phlegmasia, we distinguish in *bronchitis* the *acute*, *chronic*, *catarrhal*, and *croupous* forms; also the *serous*, *mucous*, *purulent*, and *fetid* forms, etc. It is designated *serous bronchorrhea* when exudation is very abundant, and *broncho-blennorrhea* when the exudation is purulent. The inflammation of the bronchioles (*bronchiolitis* or *capillary bronchitis*) is of particular importance. Villous bronchitis is very rare. *Verminous* and *mycotic* bronchitis constitute special forms from an etiological point of view.

Etiology of acute catarrhal bronchitis. Acute bronchial catarrh, which is particularly frequent in the horse, is also met with in the dog and ox. The inflammation may be limited to the mucous membrane of the bronchial tubes, or it may extend to the trachea and larynx; sometimes it ascends as far as the nasal cavities; in which case the affection is known as *catarrh of respiratory tracts*. Its causes are variable:

1. As predisposing conditions may be mentioned young age, a weak constitution, a lymphatic temperament, an imperfect nutrition, over-heated stables, etc.

2. Cold plays a leading rôle. In most cases it acts directly upon the bronchial tubes through the medium of inspired air. Damp weather, sudden changes in temperature, cold winds, and thick fogs

¹ Degive: Annal. de Bruxelles, 1881.

² *Tumors* of the larynx, those developed in its neighborhood, and *fractures* of the cartilages of this organ are extremely rare accidents, and belong to the domain of surgical pathology. Neoplasms localized in the neighborhood of the upper orifice of the larynx produce troubles of deglutition, dyspnoea, attacks of suffocation, and generally cause death, either by asphyxia or by producing a traumatic pneumonia. According to Goubaux, cysts of the base of the epiglottis are the result of an obliteration of the excretory canal of one or several salivary glands which exist in quite large numbers in this region.—N. D. T.

may also determine acute bronchial catarrh. Its endemic appearance in the spring and fall is explained by the action of these causes.

3. Mechanical and chemical irritations, inspiration of gas and acrid vapors, hot air and smoke, air loaded with mould, fodder which has heated or is covered with rust and black rust (Maile, Berndt). The penetration into the bronchial tubes of medicinal agents administered in the form of drinks, etc., may determine bronchitis. It is not known whether the introduction into these tubes of the pollen of grasses which are in flower may not produce it in the same way as hay-fever is caused in man. (Concerning the pathological influence of parasites, see Verminous Bronchitis.)

4. The disease occurs quite frequently by the extension to the bronchial mucous membrane of a catarrh which is at first localized in another region of the respiratory tract.

5. Sometimes bronchitis is developed which cannot be assigned to the action of cold; it is marked from the onset by intense fever. In these cases it is produced by infectious elements.¹

6. Finally, acute bronchitis occurs *secondarily* as an epiphenomenon of several infectious diseases; then it may constitute the predominating disease (disease of young age, contagious pneumonia, influenza, glanders, anthrax, variola, etc.). For this reason it is not always easy to distinguish *secondary infectious bronchitis* from *primary bronchitis*; both, in fact, may be accompanied by high fever, and pneumonia from the beginning may evolve with symptoms of bronchitis. When pyretic bronchitis is present we should always take into account the possibility of the affection developing in the course of some infectious disease. The old name "catarrhal fever" was applied to these complex cases, the nature of which is frequently difficult to determine.

Pathological anatomy. The essential alterations are tumefaction, redness, and ecchymoses of the mucous membrane; its surface is covered with serous, mucous, or purulent matter, which is especially produced by the transuded serum. The microscope shows epithelial desquamation, destruction of the mucous glands, more or less abundant emigration of leucocytes, and an infiltration of the mucous membrane and subjacent connective tissue by numerous embryonal cells. When the inflammation extends to the bronchi

¹ Bronchitis, like laryngitis, exists at times in an epizootic state. Observations upon infectious bronchitis have been given by several foreign authors and by Joly (see Recueil Vét., 1888).—N. D. T.

and alveoli, the pulmonary parenchyma participates in the morbid process, and we find alterations of catarrhal pneumonia.

Symptoms. Acute bronchial catarrh usually begins with a rather high fever (39.5° to 41.5° C.). In the horse, out of seventeen cases of acute bronchitis, in fifteen the fever reached its maximum the first day; in four instances the temperature rose above 41° , seven times it went above 40° , and four times it exceeded 39.5° . The circulation is always accelerated. We frequently observe chills and great weakness; prehension of food, rumination, and lacteal secretion disappear. The *cough* is at first dry and painful; later it becomes loose and is accompanied by a serous, mucous, or purulent discharge. On auscultation we perceive in the beginning a loud, coarse, vesicular murmur; later it is accompanied by dry *râles* (wheezing, rattling, snoring noises), which become moist as soon as the secretion is abundant; these *râles* constitute a characteristic symptom of bronchitis. As long as the disease is simple, not complicated by catarrhal pneumonia, percussion will not reveal any abnormal phenomenon. In *capillary bronchitis* dyspnoea is intense and attacks of suffocation are sometimes observed. This form is particularly noticed in very young or old animals, and in the latter it is usually complicated with pulmonary oedema. According to Saint-Cyr, the gravity of bronchiolitis is in inverse proportion to the size of the patients.

The duration of acute bronchial catarrh is on an average from two to three weeks, but frequently it does not exceed four to eight days. Except in young or very old animals, its course is generally regular and its termination favorable. A complete cure is almost always obtained. In serious cases, however, and when its causes continue to act, acute bronchitis often passes into a chronic state.

Treatment. Use fumigations of plain tepid water, or add to it cresol, phenic acid, alum, tannin, essence of turpentine, etc.; these agents are useful in all cases. Among the internal medicinal agents hydrochlorate of apomorphia is a most efficient expectorant. In the dog it is given in a daily dose of 0.005 to 0.01 gramme dissolved in 50 grammes of water; a tablespoonful of the solution should be given every three hours. Ammonia, which was formerly used systematically, is only advisable in dry cough or when the catarrh is already advanced and the fever slight; the dose is 8 to 15 grammes for the horse, 0.2 to 1 gramme for the dog. In strong animals, and when the fever is high, writers on this subject advise

tartar emetic or the sulphide or sulphuret of antimony (horse, 2 to 5 grammes; dog, 0.05 to 0.2 gramme). Ammonia and tartar emetic may be given in the form of a solution; for the dog ammonium chloride and licorice each 5 grammes, tartar emetic 0.5 gramme, distilled water 300 grammes; a tablespoonful or teaspoonful to be given three times per day. Excessive bronchial secretion is combated with essence of turpentine (for the dog, 0.1 to 0.5 gramme), senega or liquor ammonia with essence of anise-seed.¹

We sometimes give at the same time senega and liquor ammonia. The following formula is for the dog: infusion of senega-root, 10 grammes to 150 grammes of water; liquor ammonia and essence of anise-seed, 5 grammes—two tablespoonfuls or teaspoonfuls a day. When the coughing spells are very painful we must resort, as in acute laryngeal catarrh, to morphine and bitter-almond water (morphine, 1 gramme; bitter-almond water, 10 grammes; water, 150 grammes—two or three tablespoonfuls or teaspoonfuls a day). If pulmonary oedema becomes alarming, use stimulants (camphor, ether, caffen, hyoscyamine, alcohol, etc.) or emetics.

It is also advisable to institute a dietetic treatment (repose, ventilation of the stables, light and easily-digested food). [In the convalescent stage of acute bronchitis it has always been my practice to put the patient on full doses of liquor arsenici et hydrargyri iodidi, which was continued until every vestige of the disease had disappeared. This powerful alterative was given with the object of preventing the laryngeal atrophy so frequently seen as a complication of this affection, due to intrathoracic disturbance of the function of the recurrent laryngeal. I have never seen roaring or whistling complicate a case so treated, and I have seen a number of cases which were not of long standing get well under the influence of this preparation.—W. L. Z.]

CHRONIC BRONCHIAL CATARRH.

Chronic Catarrhal Bronchitis.

Etiology. Weak animals, especially young dogs and cachectic sheep, are predisposed to chronic bronchial catarrh. Among its direct causes we must first consider those which have been indi-

¹ The formula of this liquor is: essence of anise, 1 part; ammonia, 5 parts; alcohol, 24 parts.—N. D. T.

cated in the chapter on acute bronchitis. In the majority of cases it is consecutive to the latter. Sometimes it occurs during the course of heart or lung diseases; it may thus complicate valvular lesions, chronic interstitial inflammatory processes of the lungs, pulmonary emphysema, etc., affections which produce a stagnation in the bronchial vascular apparatus (the bronchial capillary plexus is joined to the pulmonary plexus). Most broken-winded horses are affected with chronic bronchial catarrh. It is also observed as an epiphenomenon of certain general diseases, such as anemia, leukemia, chlorosis, chronic Bright's disease, etc. (Concerning the pathologic action of parasites, see Verminous Bronchitis.)

Pathological anatomy. The bronchial mucous membrane is colored dark reddish-brown, and its vessels are dilated; it is thickened and sometimes covered with polypoid growths; its secretion is mucous or purulent, often putrid and fetid (fetid bronchitis); it fills the small bronchial tubes entirely, and produces *atelectasis* of the corresponding pulmonary lobules, a state which may be complicated by chronic interstitial inflammatory and catarrhal processes. When the inflammation is of long standing the various layers of the bronchial walls (mucous membrane, true bronchial tube, and peribronchial tissue) are thickened (chronic endobronchitis, mesobronchitis, and peribronchitis). This alteration is the consequence of a connective-tissue neoformation, which is preceded by an infiltration of embryonal cells. Under the name of *nodular peribronchitis*, Dieckerhoff has described a particular form of peribronchitis of the horse. It consists of a multitude of small nodes of the size of a millet-seed or that of a pea; they are hard, whitish-gray when cut, and the contents are sometimes formed of caseous or calcified bronchial secretions, which give them a great resemblance to glandered tubercles. They are distinguished from these latter, however, by their uniform size and the similarity of age; besides, we may easily detach and extract their central part. Nocard has contested the exactness of these anatomo-pathologic peculiarities. According to Ziegler, peribronchitis which extends to the bronchioles must naturally be related to pneumonia; this author designates it under the name of follicular broncho-pneumonic induration.

One of the most frequent consequences of chronic bronchial catarrh is *bronchiectasis*; that is to say, a dilatation of the bronchial walls, which are rendered less resistant by the catarrhal process;

this is an alteration which is especially observed at the apex of the pulmonary lobes in the horse.

We distinguish cylindrical, sacciform, and fusiform bronchiectasis. The dilated canals are filled with mucus and thick decomposed pus, which is oftentimes calcified (pulmonary calculus); these calcareous masses are of the size of a pea, or may be as large as a chestnut or the fist. The small bronchi frequently reach the size of a finger.

Emphysema is also frequent in chronic bronchial catarrh.

Symptoms. These differ from those of acute bronchitis only in the absence of febrile reaction. The principal symptom is cough, which is, as a rule, accompanied by an abundant mucous discharge. On auscultation we hear dry and moist râles of various kinds, which are disseminated through both lungs. When chronic bronchial catarrh is accompanied by atelectasis and emphysema, dyspnoea is intense, and we observe all the symptoms of broken-wind. Later, nutrition is impaired. The animals become emaciated and gradually weaker, fall into cachexia, and when the disease is neglected often die from catarrhal pneumonia.

Bronchiectasis is characterized by a decomposing, muco-purulent fetid discharge, which is sometimes very abundant. Percussion of the costal walls gives a tympanitic resonance.

Treatment. The cure of chronic bronchial catarrh is only obtained by long-continued treatment. The means which should properly be resorted to differ very little from those indicated in the acute form. Expectorants and stimulating agents are indicated: essence of turpentine, tar, cresol, ammonia, etc.; they may be administered internally or in fumigations. Intra-tracheal injections, which have been recommended quite recently by Levi (1889), were formerly tried by Lafosse (1854). They are dangerous and of doubtful efficacy. In man the treatment of bronchiectasis is partly surgical.

It has been advised to combat chronic bronchial catarrh by physostigmine and pilocarpine. We must warn practitioners of accidents to which the use of these alkaloids exposes animals. A vigorous horse, which was affected with chronic bronchial catarrh, and to which we gave a subcutaneous injection of 0.1 gramme of eserine, was taken with intense dyspnoea and died from asphyxia within three-quarters of an hour. At the autopsy we found a diffuse pulmonary œdema. We have also seen dogs affected with

chronic bronchitis sink rapidly after a hypodermatic injection of 0.05 gramme of pilocarpine.

BRONCHIAL CROUP.

Croupous Bronchitis.

Etiology. Bronchial croup, which is quite frequent in the ox and sheep, is due to the same causes as croupous laryngitis: colds, sudden changes of temperature, inhalation of smoke in case of fire (Rey, Jensen), also penetration into the trachea of irritating medicinal agents. We may also observe it as an epiphenomenon of croupous pneumonia and of certain infectious diseases (bovine plague, malignant catarrhal fever, contagious peripneumonia, etc.). Mayrwieser has described an infectious croupous bronchitis in the ox.

Pathological anatomy. The mucous membrane of the trachea and bronchial tubes is injected, tumefied, and covered with a thick, yellowish membranous exudate, half a centimetre in thickness, smooth upon the surface, and but slightly adherent to the mucous membrane, which has an anatomical composition similar to that of the exudate of croupous laryngitis. Sometimes we find on the posterior part of the trachea lamellar exudates which are rolled up. The small bronchial tubes are frequently obstructed by cylindrical, yellowish, elastic masses similar to thrombi. The lungs are hyperemic. In the other organs we recognize the alterations of asphyxia.

Symptoms. In some cases the disease begins with slight general symptoms, such as weakness, inappetence, and cough; in others, which are more frequent, it is characterized from the onset by alarming disturbances. In general, an intense dyspnoea is the first symptom noticed; respiration is accelerated, and there is rattling, snoring, or wheezing; the cough is spasmodic and painful. We observe at times expectoration of cylinders of croupous membranes which may reach a length of 50 centimetres (Fraüenholtz). During auscultation of the trachea and bronchial tubes we detect many râles. The hand applied on the trachea is sensible of a manifest quivering. Percussion of the chest does not reveal anything abnormal. Soon we observe symptoms of asphyxia: cyanosis of the visible mucous membranes, very laborious breathing, gaping mouth, extended tongue, anxious look, etc.

Croupous bronchitis usually reaches the period of greatest intensity toward the second or third day, then its course is rapid toward resolution or death by asphyxia. In the first case an abundant expectoration of false croupous membranes is produced. Death is the ordinary termination in young animals and those having a narrow trachea. The duration of the disease does not exceed six to eight days.

Treatment. The very rapid evolution of croupous bronchitis renders the treatment of little avail. We may try to soften the exudate by inhalations of steam or of the vapor produced by slaking quicklime. In small animals it is advisable to administer an emetic. In the bovine species the cure of seriously-affected subjects must not be attempted.

VERMINOUS BRONCHITIS. VERMINOUS DISEASE OF THE LUNGS.

Etiology. Verminous disease of the lungs, which is also called verminous cough, is caused by strongyles which are lodged in the trachea and bronchial tubes, where they set up successively verminous bronchitis, catarrhal pneumonia, and finally "verminous phthisis."

The strongyles which are found in domestic animals are :

1. In the SHEEP (and goat) *Strongylus filaria*, more rarely *Strongylus paradoxus*, and *Strongylus rufescens*, exceptionally *Pseudalius ovis pulmonalis* of Koch ;

2. In the OX, very rarely in the horse and ass, the *Strongylus micrurus* ;

3. In the PIG the *Strongylus paradoxus* ;

4. In dog, cat, and rabbit, several species of which as yet little is known ;

5. In birds the *Syngamus trachealis* (see Diseases of the Respiratory Apparatus of Chickens).

In the hare the *Strongylus commutatus* (Kitt, Mégnin, Rémy) produces verminous centres of pneumonia with calcification, which present a striking analogy to tubercular lesions.

NATURAL HISTORY. When in a sexual condition the strongyles are located in the trachea and bronchial tubes of the sheep, ox, and pig. Eggs and embryo are developed in the ducts ; those which are expelled by coughing continue to grow in pools and swamps. We do not yet know if their complete evolution requires an inter-

mediate host (Insect, Mollusc). The larvæ are ordinarily ingested with water and food. Zürn admits the possibility of their introduction into the bronchial tubes by the inhalation of dust in which they are suspended.

Infection of the animals usually occurs in spring; according to Gerlach, it does not take place when the crops have been harvested. The disease is dormant for some time; the diseases produced by strongyli do not appear before the fall. It is very common during rainy years, but may disappear altogether during dry years. Bauer has seen it appear in the Alps after a wet summer, where it had not been observed for eight consecutive *dry* years. This fact clearly establishes the influence of dampness upon the development of strongyles. In certain damp and swampy countries the verminous disease is stationary and enzootic. It has never been recognized in animals kept permanently stabled.

Direct contamination is scarcely possible. It is likely that the embryo after having entered the stomach reaches the bronchial tubes by the œsophagus and trachea.

The tenacity of existence of the strongyles and their embryos is great. Researches of Colin have demonstrated that the embryos which escape from cadavers containing strongyles preserve their vitality in water for more than two months. According to Ercolani, the parasites resist exposure to heat for thirty days and also the action of alcohol and sublimate solution (1 per cent.) when prolonged for eight days.

Enzootic verminous bronchitis of sheep often causes considerable damage. In Algiers, on the frontier of Morocco, there are years, according to Carnet, when this pest destroys one-third of the flocks. Its gravity has caused it to be classed in certain countries among diseases which are considered as setting aside a contract of sale. (In Austria it may set aside a contract of sale after a period of two months.)

Pathological anatomy. The alterations found in the bronchial tubes and pulmonary tissue are very variable. We observe at times lesions of chronic bronchitis and bronchiectasis, oftentimes lobular centres of broncho-pneumonia, in some instances neoformations of tuberculous appearance (pseudo-tuberculous pneumonia or nematoid tuberculosis). According to Bugnion, we find in the goat diffuse pneumonia which is produced by the eggs and embryo of the strongyles.

1. Verminous bronchitis is characterized anatomically by a considerable *tumefaction* and a *purulent* or *hemorrhagic phlegmasia* of the bronchial mucous membrane; this is covered with nodes containing the parasites. In the trachea and bronchial tubes are found balls formed by worms and coated with mucus and pus; these ducts present sacciform dilatations at these points. The interior of the bronchial tubes is filled with a purulent viscous exudate which in some instances is hemorrhagic, and in which may be found mobile eggs and embryo. In the neighborhood of the bronchial dilatations the pulmonary tissue is thickened, atelectatic, splenized, or emphysematous; the lungs are œdematous or anemic.

2. Besides these lesions we may observe *centres of lobular pneumonia*. These are produced by an extension of the inflammation to the pulmonary tissue, and consist of hepatized islands, on the surface of which later appears an atelectasis, splenization, or purulent infiltration. These alterations are particularly found in calves and pigs; they have their starting-point in the balls of adult strongyles. In the pig the parasites locate preferably toward the apex and margins of the lungs. According to Kohlhepp, we may suspect their presence when, on a superficial examination, the upper part and edges are larger than usual, or when certain lobules do not become depressed as in a normal state.

3. *Pseudo-tuberculo-pneumonic centres* represent tuberculiform neoformations of various dimensions, the pulmonary tissue of which is often invaded to such an extent that they are easily to be perceived upon the surface of the borders of the organ; they frequently render its surface irregular and uneven, and lead to pleural adhesions. These neoformations are formed by nests of worms, and are surrounded by a connective-tissue capsule(?), upon incising which there escapes a mass consisting of pus and eggs of strongyles; these are small peribronchial centres produced by inflammation extending from the bronchial tubes (Van Tright).

Besides pulmonary alterations we recognize in serious cases hydremia, dropsies in the several cavities and in the connective tissue, and all the symptoms of anemia. In the pig we find tumefaction of the mesenteric, peri-laryngeal, and retro-pharyngeal connective tissue.

The disease which is described by Koch under the name of *disease of pulmonary hair worms* (*Lungenhaarwurm krankheit*) is due to *Pseudalius ovis pulmonalis* (Koch). The nematode was observed

by Brown in 1851; later by Cobbold and Axe; Gray, Sandie, and Padley have given it the name of *Nematoideum ovis pulmonales*; ¹ more recently Utz, Lydtin, and van Tright have studied this affection. Railliet considers *Pseudalius* as identical with *Strongylus rufescens*. Motz found it in $4\frac{1}{2}$ per cent. of the sheep killed for consumption; the parasites seem to be equally numerous during all seasons of the year. The alterations in the lungs are just as variable as in strongylosis; with the naked eye it is possible to recognize two different forms of the pseudo-tuberculo-pneumonic centres:

1. In the one form they are small, thickly crowded, and of the size of miliary tubercles; their color is yellowish-white; they contain small calcified worms, which may be recognized with the naked eye. A microscopic examination shows that they are formed by an accumulation of white corpuscles, in the centre of which exist the twisted filiform nematodes. On the periphery of these small inflammatory centres we find a capsule of connective tissue.

2. The other form shows dimensions varying between those of a lentil and those of a nut; they are distinctly defined, of a yellow or grayish-yellow color; sometimes soft and elastic, at other times hard. On section they are found to be filled with a milky, frothy liquid, containing small filiform worms, some of which are brown (males), others white (females). The larger contain only caseous elements. The development of these nematodes appears to be identical with that of strongyles; the diseases which they produce are also similar.

Symptoms. The symptoms of verminous disease are those of serious bronchial catarrh with consecutive "pulmonary phthisis." Castrated sheep and lambs are mostly affected by it. The animals begin to cough, which is at first strong, croaking, rattling, later very weak; it is excited when the animals are exercised; it is often observed in all the subjects belonging to a flock. We observe besides a mucous discharge, salivation, nausea, and ejection of worm balls; the animals frequently rub the nose upon the ground. Respiration is laborious, wheezing, rattling; on auscultation of the trachea and bronchial tubes we perceive a great variety of râles. Later dyspnœa becomes intense; the patients are very weak and take their food kneeling. The skin and mucous membranes become very pale; the wool loses its stiffness. In some cases death occurs through exhaustion after weeks or months, but asphyxia

¹ Gray, Sandie, and Padley: Annals and Magazine of Natural History, t. ii.

may determine it sooner. According to the state of nutrition and alimentation the mortality varies between 10 and 70 per cent.

In the ox the symptoms are similar; here again the disease is only observed in young animals, from one to one and a half years old. Sometimes the manifestations of verminous pneumonia excite a suspicion of the existence of peripneumonia or tuberculosis. As a general rule the ox is much more resistant than the sheep. Quite recently, during the course of an epizootic of strongylosis (*Strongylus micrurus*) Claes saw in almost all the sick oxen an ophthalmia which was occasioned by the presence of the *Filaria papilosa* (?) in the anterior chamber of the eye. It is rare to recognize the disease in animals which are kept in the stable.

In the pig the *Strongylus paradoxus* seems to be quite common; but this animal is even less sensitive than the ox to the action of this parasite. Nevertheless if strongylosis is rare in the adult pig, it is frequent and of grave import in young pigs, in which it often exists in an enzootic state. The animals are affected in varying degrees; we may notice cases of sudden death produced by pulmonary oedema; this termination is not so rare as is maintained by some authors. With the development of the animal the disease improves materially.

Strongylosis is very rare in the other species. In a cat Colin observed small pulmonary tubercles of yellowish color, which varied in size from a millet-seed to a hempseed, and which contained strongyles. He admits that the cat becomes infected by eating ox or sheep lungs, a food which is the usual fare of this animal in Paris. Eberth has also found nematodes upon the surface of the lungs of cats; the smooth muscular fibres (Reisessen's muscle) were considerably hypertrophied. In the hare Prietsch, Mégnin, and others have found the *Strongylus commutatus*. Finally, in the dog Blumberg and Rabe have found in the trachea and bronchial tubes a filiform nematode which had determined on the mucous membrane small warty pimples of the size of a millet-seed to a grain of coffee (warty rhachitis).

Treatment. The prophylaxis of verminous bronchitis consists in avoiding wet pastures in the spring of the year. This measure, however, sometimes presents almost insurmountable difficulties.

The disease is combated by fumigations of tar, phenic acid, cresol, essence of turpentine, tobacco, etc. The practice of fumigating is very simple: the animals have to be kept for a variable time in a place

which is filled with the vapor of one of these agents; the vapors, by irritating the respiratory passages, produce coughing and expulsion of the worms. We must be careful to destroy the parasites which are thus ejected. It is also advisable to give to the animals easily digestible food and to resort to ferruginous preparations. Lately intra-tracheal injections of anti-parasitic medicines have been advised: phenic acid, essence of turpentine, cresol, chloroform (Levi, Eloire, Chelchowsky, Claes, Neimann, etc.). Eloire uses the following preparation: olive oil, 100 grammes; essence of turpentine, 100 grammes; phenic acid, 2 grammes; fetid animal oil, 2 grammes; he injects it into the trachea in a dose of 10 grammes per day for three consecutive days. We may utilize in the same way a mixture of oil, essence of turpentine (— 100 grammes), and cresol (5 grammes).

V. DISEASES OF THE LUNGS.

GENERAL CONSIDERATIONS ON PHYSICAL EXAMINATION OF THE LUNGS BY PERCUSSION AND AUSCULTATION.

Physical Diagnosis of Pulmonary Diseases.

Without percussion and auscultation a diagnosis of diseases of the lungs is impossible. The limits of this work do not allow us to make a complete study of these methods of exploration; we must refer to special works, and recommend particularly the *Treatise upon the Clinical Methods of Examination*, by Guttmann. Notwithstanding that this book has been written by practitioners of human medicine, veterinarians will find much useful information in it.¹

PERCUSSION OF THE LUNGS.

HISTORICAL. Percussion of the thorax, which is performed with the object of establishing a diagnosis of diseases of the lungs, was introduced by Aüenbrugger (1753), and afterward studied in the beginning of this century by Corvisart, Piorry, and Skoda. Skoda invented the pleximeter (1826) and described the tympanitic sound

¹ The practical manual on exploration of the chest in domestic animals, by Prof. Saint Cyr, is an excellent work, in which everything concerning percussion, auscultation, and pneuography is very completely and clearly described.—N. D. T.

(1839). In 1841 Wintrich had the pleximetric hammer constructed. Among modern authors who have written on this subject we may mention Traube, Niemeyer, Gerhardt, and Guttmann. Percussion was applied in veterinary medicine by Dupuy (of Alfort), in 1824, and was studied later by Röhl (of Vienna).

METHODS OF PERCUSSION. We distinguish *immediate* and *mediate* methods of *percussion*. *Immediate percussion* consists in practising upon the thorax slight raps with the hand or with the bent finger; this method is no longer used. In small animals it may serve to recognize a certain painful condition of the lungs and plexus, which is detected by the subjects coughing at the time of percussion.

Mediate percussion is performed by interposing between the percussing finger and the thoracic walls one or several fingers of the free hand; these latter may be replaced by the pleximeter—that is to say, by a small, thin, elastic cover. The *pleximeter* should be non-resonant; the material best adapted for its construction is ivory; the use of metallic covers should be avoided. Percussing *finger upon finger* is useful in small animals and upon irregular thoracic surfaces; it permits us to map accurately the healthy pulmonary tissue and the diseased parts; moreover, the *sensation of resistance* is much more clearly indicated than by the use of the pleximeter. After examining a certain number of patients the fingers begin to smart; we must then resort to the pleximeter and percussion hammer.

OF THE SOUNDS PRODUCED BY PERCUSSION. In percussion the degrees of intensity of sound are not designated under the names *strong* and *weak*, *acute* and *grave*; the terms *clear* and *dull* are used, and we express by the word *subdulness* (*gedämpft*—subdued) the degree of resonance lying between these two extremes. Skoda used the terms *full*, *dull*, and *empty*. The intensity of sound depends, on the one hand, upon the consistency and thickness of the pectoral wall, and, on the other, on the abundance of air in the lung. Percussion of the throat, which is very muscular, gives a sound which is relatively dull; this character is especially marked in the pectoral regions of the great dorsal. In very muscular and fat horses percussion may elicit subdulness—even dulness—over the entire area of the thoracic walls. The dulness increases as the quantity of air contained in the lung diminishes; when the pulmonary parenchyma is completely emptied of air dulness is complete (*muscular* sound, *femoral* sound).

INFLUENCE OF PULMONARY DISEASES ON PERCUSSION BRUITS.

Percussion gives, or may give, a dull sound in pneumonia which has reached the stage of hepatization, in pulmonary atelectasis, in interstitial thickening of the lungs, pulmonary or pleural neoformations (tuberculosis), and in compression of the lungs by liquid which has accumulated in the thorax. Diseases of the bronchial tubes, without participation of the pulmonary tissue in the process (acute and chronic bronchitis), do not influence the bruit of percussion. Pulmonary œdema sometimes produces subdulness, at other times a tympanitic bruit.

TYMPANITIC SOUND. This differs from the clear normal percussion bruit by its ringing sound, which is never observed in a normal thorax; we also further designate the normal sound by the expression *non-tympanitic*. For comparison it may be elicited by percussing the abdominal viscera which contain air. Like the drum sound, from which its name is derived, it is produced by the reflection upon smooth walls of vibrating air. Notwithstanding that this condition may be fulfilled in the pulmonary alveoli, the tympanitic sound is not produced on percussing the normal thorax, because in the latter the lung is always distended in excess of its physiological volume; percussion produces a vibration of the pulmonary tissue and alveolar walls with the air which is contained in the lobules. It is for this reason that the air-waves are not reflected regularly; these waves, which are from various sources, become mixed and confounded, and the normal sound of percussion thus produced is not musical. Percussion of the flabby and relaxed lungs (coming from the thorax) gives a tympanitic bruit; it is sufficient to inflate the organ in order to produce a return of the clear, normal sound. The same phenomena may be recognized by percussion of the intestine.

The percussion sound of the thorax is tympanitic:

1. When cavities into which the air has access are formed within the pulmonary parenchyma (bronchiectatic caverns); but these cavities must be spacious enough and almost closed, and surrounded by solid walls (dense connective tissue), which is favorable to a reflection of sonorous waves.

2. In pneumothorax. The air contained in the thoracic cavity is under a certain pressure, and the smooth walls of this cavity reflect the air-waves.

3. When the tension of the pulmonary parenchyma is diminished

consecutively to a serous infiltration (pneumonia in the stages of augmentation and resolution, pulmonary œdema, slight compression of the lungs by the pleuritic exudate); in this latter case we frequently recognize a tympanitic sound above the line of dulness.

CRACKED-POT SOUND. Also designated by the expression *metallic tinkling*. [This bruit has a peculiar character of sound, caused by the sudden expulsion of air from a cavity through a small opening in its walls, and it is well heard when the hands are pressed together and struck upon the knee in a manner well known to schoolboys, so as to produce a noise closely resembling the rattling of coin. It derives its name (which Laënnec first gave it) from the resemblance to the sound produced by striking a cracked jar.¹—w. L. Z.] It is generally pathognomonic of the existence of a cavity, and is produced by the air which passes through the latter into a bronchial tube. (When the pleximeter is not accurately fitted to the thoracic wall the air which escapes from below the plate at the time of percussion produces also a cracked-pot bruit.) Metallic tinkling is also observed in cases where a pleuritic exudate mixed with air is confined in an abnormal cavity formed by joining of the pleural layers (contagious pneumonia of the horse), and in pneumothorax when consecutive to a pleuro-pulmonary perforation or produced by a penetrating wound of the chest; lastly, it may be seen in the course of pneumonia in the neighborhood of the hepatised or necrosed pulmonary tissue. We may also recognize cracked-pot bruit on percussion of the thorax when the neighboring regions are the seat of a subcutaneous emphysema; it suffices to be advised of it in order not to confound this accident with pulmonary cavities.

METALLIC ECHO OR AMPHORIC SOUND. This is similar to the tympanitic sound in its characters, but its resonance is more prolonged. It is produced in cases where the lungs contain large cavities, in greatly accentuated pneumothorax, and sometimes at the period of resolution in pneumonia when the flabby and relaxed alveoli contain air in abnormal quantity.

RESISTANCE TO THE FINGER OR TO THE PLEXIMETRIC HAMMER OF THE PERCUSSED PARTS. This resistance is greater in proportion as the percussed regions are more dense, more deprived of air, and less elastic. It is more marked, for instance, when a considerable proportion of liquid is accumulated in the pleura and

¹ Brown: Medical Diagnosis, p. 159.

in cases of pneumonia ; this symptom may be used for a differential diagnosis of these two diseases.

TOPOGRAPHY OF THE THORAX FROM THE STANDPOINT OF PERCUSSION. 1. In the *horse* we divide the costal wall of the thoracic cavity into nine equal parts by means of two vertical lines and two others which are transverse. The central square and part of the upper and lower median squares are alone explorable by percussion. In regions of the upper zone we always recognize subdulness, which is due to the dorsal muscles (great dorsal, ilio-spinal, etc.); those of the anterior zone are covered by the shoulder and arm, and the muscles of the posterior zone correspond to the abdominal viscera. On the left side of the inferior zone, between the third and sixth ribs, the precordial region exists ; by means of a half-moon shaped furrow of the left lobe the heart rests there directly against the costal wall. Toward the lower part of the thorax and between the fourth and sixth ribs upon the left side, we find a dull region which corresponds to the heart. 2. In ruminants the thoracic cavity is shorter than in the horse, because in these animals the ribs are less numerous and the diaphragm more convex in front. *a.* In the *ox*, in the upper third of the thorax, the normal pulmonary sound extends as far as the tenth rib ; behind this point we find, to the right, loops of the intestine ; in the third median the normal pulmonary resonance extends to the ninth rib ; further, we find upon the left the rumen, on the right the liver. The half-moon shaped furrow of the left lung is wanting ; nevertheless the heart may be explored by striking hard. *b.* In the *sheep* the field of percussion is very circumscribed ; it is limited by the posterior border of the olecranon muscles, by a horizontal line on the upper third of the thoracic cavity as far as the tenth rib, and by another line starting from this point of the tenth rib to the lower extremity of the olecranon muscles. 3. The thorax of the *dog* is easily percussed ; it may be examined in its sternal region and in its upper third ; in this animal the pericardium adheres to the diaphragm, and the heart is situated in the median plane of the body ; on percussing vigorously we obtain, nevertheless, *cardiac sounds*. 4. In other animals percussion is of secondary importance. In the *pig* it is particularly difficult by reason of a thick layer of adipose tissue.

In a general way, we may say that this method of exploration furnishes in animals diagnostic information which is less complete than in man, because the anatomical conditions and the relations of

the shoulder and thorax particularly restrict the field of percussion. We must also take into account the difficulties that certain animals offer to an operation, either by their misbehavior or on account of the anxiety to which they are subjected when being examined.

AUSCULTATION OF THE LUNGS.

HISTORICAL. Hippocrates mentioned certain abnormal thoracic bruits; among others, the splashing sound of empyema; but the enunciation of the principles of auscultation is due to Laënnec (1816). Skoda, P. Niemeyer, and Baas have taken up and completed his studies. In France auscultation was first applied to domestic animals by Dupuy, Leblanc, Delafond, Hurtrel d'Arboval, H. Bouley, etc. Among German veterinarians who have used this method of exploration we may mention Hofacker, Müller, Röhl, Gleisberg, Postl, and Vogel.

METHODS OF AUSCULTATION. We distinguish *immediate auscultation* and *mediate auscultation*. In the first the ear is applied directly to the pectoral walls; in the second we use the stethoscope. This instrument is not essential in auscultating animals.

BRUIES WHICH ARE RECOGNIZED IN AUSCULTATION. We must distinguish: 1, purely respiratory bruits—that is to say, *vesicular*, *bronchial*, and *vague respiratory bruits*; 2, *râles* due to the presence of liquid products in the bronchial tubes; 3, *bruits of friction* occasioned by a sliding of the roughened pleural surfaces upon each other. The first two are normal; on the trachea we perceive tracheal and bronchial respiratory bruits; in the lungs we recognize the vesicular bruit. *Râles* and friction bruits are always abnormal.

VESICULAR RESPIRATORY BRUIT. This is a normal pulmonary bruit, and is perceived when the inhaled air penetrates into the *infundibula*; when the entrance of air into this pulmonary lobule is prevented by exudates the vesicular murmur disappears. It has been considered as a bruit of friction or of oscillation. According to the theory of Baas, which appears to us to be the best founded, the vesicular murmur is but a continuation as far as the lung of a modified laryngeal bruit. This latter is the bruit of a contracted tube (glottis), which is extended into the trachea and large bronchial tubes. Its principal character is a sound which suggests a pronunciation of the letters "ch." When the sonorous waves of the laryngeal, tracheal, or bronchial bruits reach the lungs they become

weak and produce a mild vibratory sound, the fundamental character of which is the "v." This theory has experimentally demonstrated: a mediate auscultation of the larynx is performed by placing upon the latter the inflated lung of an animal—we do not hear any laryngeal or bronchial bruit, but a vesicular murmur; by covering up the throat with a piece of leather we perceive distinctly a laryngeal bruit. When normal the vesicular murmur, which is very mild, is only perceptible in animals when at rest; it becomes rough and hard in bronchial catarrh, and when there is a diminution in the breathing surface of the lung (supplementary respiration). In young animals the vesicular murmur is hard and coarse on account of the greater elasticity of the pulmonary tissue and of the thinness of the pectoral walls ("puerile" vesicular murmur which exists in the boy up to the twelfth year).

BRONCHIAL RESPIRATORY BRUITS (*Laryngeal or tracheal bruit*). This bruit is caused by a contracted tube. It has a blowing character, which may be normally perceived upon the larynx, along the trachea, and upon the surface of the bifurcation of this duct. Its existence in the pulmonary lobes (tubal blowing) is pathological. It is found: 1. In cases of pulmonary cavity and bronchiectasis when these lesions are not too deeply located and are of considerable dimensions; besides, these cavities must possess thick, rigid walls and be freely joined to the large bronchi (a very rare peculiarity); 2, when the pulmonary tissue is deprived of air, either by hepatization or by compression exerted upon it by liquids or air. In this case "the bronchial respiration" is due to the propagation of the laryngeal bruit by the collapsed lung. Trasbot has given a demonstration of the appearance of the bronchial bruit in the lung when this organ no longer contains air. This author made a section of the trachea of a horse suffering with pneumonia in which a very manifest bronchial blowing was observed; by bringing the inferior end of the trachea outside the blowing would disappear instantly, because the laryngeal bruit was no longer transmitted to the lung; by joining again the ends of the trachea the blowing would immediately reappear. It is also possible that the bronchial respiration may be due in part to the oscillation communicated to the air contained in the bronchial tubes by the respiratory current which passes over their mouths; this mechanism is identical with the whistling noise produced by blowing over a hollow key placed to the lips.

Amphoric blowing is a variety of the bronchial respiratory bruit; it is characterized by a metallic sound similar to that obtained by blowing into a pitcher. It is observed in pulmonary cavities of large extent which communicate freely with a bronchial tube; they may also be present in pneumothorax.

VAGUE RESPIRATORY BRUIT. This bruit presents neither the character of the vesicular nor that of the bronchial bruit; it has as a type the expiration bruit in the healthy animal at rest. It is produced by the following three factors: a decrease in the quantity of air entering the lungs, a diminution in the elasticity of this organ, and a partial displacement of the normal respiratory bruit by accessory bruits. It is observed: 1, when the pulmonary alveoli are insufficiently dilated on account of compression, atrophy, and serous or inflammatory infiltration of the lungs, as in pulmonary emphysema; 2, in cases of partial obstruction of the bronchial tubes; 3, when stronger bruits, and especially râles, are present at the same time.

RALES. These are always abnormal, and are observed in diseases in the course of which the bronchial mucous membrane is inflamed, and when liquids are excreted on its surface in a greater or less quantity. The mucous membrane is tumefied and covered with irregular wrinkles, which are disposed in a variety of ways. In the large bronchial tubes the liquid products form bubbles, which burst, producing *large bubbling râles*; the mucus which is set in motion by the air also occasions râles; in the small bronchi medium or small bubbles may burst, producing *medium or small bubbling râles*. Finally, the bronchial and alveolar agglutinated walls, becoming forcibly separated by the air current, produce *crepitant râles* (crepitation râles, with small and uniform bubbles). Crepitation is a symptom of pneumonia in its first and last stages, also of bronchiolitis, pulmonary œdema, and compression of the lungs.

According to their characters, we divide râles into *dry* and *moist*. Dry râles (creaking of new leather) present certain forms. *Crackling sounds* are produced by separation of the walls of the agglutinated bronchi; the *buzzing* or *humming bruits* (*sonorous bronchi*) are the effect of the vibration of lamellar masses in the large and medium bronchial tubes. *Whistling* bruits and *sibilant* bruits are produced in the small bronchi and bronchioles, which are partially obstructed and reduced to narrow cracks; they bear also the name

of *catarrhal* râles; they are frequently recognized during the course of acute or chronic bronchitis. *Quivering* and *singing* or *sonorous* bruits are especially found in emphysema. In pulmonary cavities the râles acquire the resonance (*sounding* bruits of Skoda), even a metallic sound; they are generally accompanied by amphoric blowing. They are designated by the name "metallic phenomena" or "cavernous phenomena."

PLEURITIC FRICTION BRUIT. This is produced when the walls of the costal and pulmonary pleura, which are ordinarily very smooth, have become rugous; it is therefore characteristic of pleurisy. In cases where the friction is very pronounced it may be perceived with the hand applied upon the costal walls (pleural quivering). It consists of dull cracklings, and a series of bruits of *friction*, of *gliding*, or of *rasping*. It is observed in the beginning of pleurisy and at the period of absorption of the liquid, especially at the termination of inspiration. The ear detects the same peculiarities as the hand. We may obtain an idea of this bruit by slightly rubbing the concha with the back of the hand.

We must be careful not to confound friction bruits with râles: the latter are very pronounced vesicular bruits or buzzing, sibilant or whistling sounds, which have nothing in common with rubbing, friction, or filing bruits; moreover, râles undergo peculiar modifications under the influence of coughing spells.

It is easy to distinguish pleuritic from pericardial frictions; the first are synchronous with the respiratory movements, and the others with the cardiac systoles.

PULMONARY HYPEREMIA AND ŒDEMA.

Etiology. 1. *Active pulmonary hyperemia* is usually observed in well-fed, plethoric animals. It is more frequent during warm weather than at other seasons of the year; it appears ordinarily after violent exertions or a rapid run. Inhalation of very cold or very warm air and acrid, irritant gases may also determine it. Vascular obliterations which are accompanied by ischemia in a large pulmonary area, followed successively by exudation, compression, etc., often produce an active *collateral* hyperemia of the lungs. These are the phenomena of active pulmonary hyperemia, which is the early stage of croupous pneumonia.

2. *Passive pulmonary hyperemia* is generally a result of cardiac

lesions complicated by blood stagnations ; to this group belong all diseases accompanied by atony of the heart which lead to hypostatic pneumonia. It is sometimes an epiphenomenon of the compression of large bloodvessels by the accumulation of gas in the stomach and intestine.

3. *Pulmonary œdema* is produced by a filtration of bloody serum into the alveoli, bronchioles, and bronchial tubes. It may be caused in various ways. *Inflammatory pulmonary œdema* represents, in fact, a serous pneumonia which is consecutive to active, intense pulmonary hyperemia occurring during the course of croupous pneumonia or some infectious diseases. Some of the cases of acute œdema of the lungs mentioned in the ox and sheep appear to be related to the infectious disease known as *malignant œdema*. In the sheep Kitt has observed acute pulmonary œdema, the cause of which could not be determined by the macroscopic alterations recognized at the autopsy, but the abundant serum which escaped from the lungs contained a great many bacilli of malignant œdema. It is possible that cases of pulmonary œdema of the ox which have been published by Nagel and Albrecht were due to that specific cause.

Stagnation of blood in the lungs frequently occasions œdema of that organ ; this alteration is produced at the time of extreme suffering, when the heart is weak and powerless to clear the lungs of the venous blood by which they are congested. Pulmonary œdema appears at times to be the result of an abnormal permeability of the vascular walls ; the acrid gases obtain entrance probably through this mechanism.

Pathological anatomy. In active pulmonary hyperemia the lungs are engorged with dark red blood ; their volume is increased and their consistency is firmer than in the normal state. The blood flows freely from the incised lung tissue ; the capillaries are dilated and prominent upon the surface of the alveolar cavities. The bronchi contain abundant frothy mucus which is more or less colored by the blood.

In passive pulmonary hyperemia we observe, at an advanced period of the disease, lesions of *brown induration (obstructed lung)*. The parenchyma is hard and studded with hemorrhages, at the site of which black pigmentary spots are later developed ; the bloodvessels are much dilated ; the mucous secretions of the bronchial tubes contain numerous red corpuscles ; in both lobes the pulmonary tissue is splenized in spots.

Pulmonary œdema is characterized by a considerable increase in the volume of the organ and by an abundance of serum which escapes from the cuts; this serum contains numerous very fine bubbles, which are also found in the bronchioles. There are, besides, a slight epithelial desquamation and an abundant migration of red corpuscles.

Symptoms. The symptoms of active hyperemia and of œdema of the lungs appear suddenly. The animals are overcome by intense dyspnea; we often count 60, 80, and 100 respirations per minute. The nostrils are much dilated; the visible mucous membranes are red; we may observe epistaxis or a frothy nasal discharge; the subjects are restless, anxious; asphyxia seems imminent. There is a slight short and dry cough. In active hyperemia percussion gives a normal resonance, but on auscultation we perceive an increased vesicular murmur. In pulmonary œdema we recognize at times a tympanitic resonance, at other times a simple exaggeration of the normal resonance; on auscultation we detect crepitant râles, and later, at the approach of death, large bubbling râles. In passive œdema (hypostatic pneumonia) there is sometimes dullness. The pulse is accelerated, full, hard, and the cardiac action is tumultuous. When the disease is likely to have a fatal ending dyspnea diminishes gradually and the animal lapses into a state of stupor which becomes more and more profound.

Course. The course of active pulmonary hyperemia is always rapid; its duration rarely exceeds twelve to twenty-four hours. It ends usually in recovery; sometimes it kills by asphyxia, or it may become complicated with pneumonia.

Prognosis generally is not grave.

Treatment. Pulmonary hyperemia is one of the rare diseases in which the abstraction of blood constitutes the principal therapeutic indication. A copious bleeding will result in cure in almost all cases. We should endeavor to produce a cutaneous derivation by means of dry irritating frictions. Laxative clysters and cold rectal irrigations are also useful means.

PULMONARY HEMORRHAGE.

Hemoptysis.

Pulmonary hemorrhage appears sometimes as an essential primary morbid state, but generally it is a symptom of very different dis-

eases. As a general rule, it is not very frequent ; it is most commonly observed in the horse, but it is not rare in the ox, as proved by the observations of Cartwright, Jacob, Guilmot, Lessona, Hezel, and Rossignol. The alarming character of its clinical symptoms has for a long time attracted the attention of practitioners.

Etiology. In the majority of cases hemoptysis takes place from the pulmonary tissue itself, more rarely from the bronchial mucous membrane, and exceptionally from that of the larynx. Its causes are variable. In the horse it may be caused by violent exertions, by drawing very heavy loads, long runs when the animals are ridden (Percivall), concussion and exhaustion of over-excited horses (Dekker). The pulmonary processes of glanders (horses) and tuberculosis (ox) quite frequently engender this condition by the suppuration and extension of the cavities, large bloodvessels are destroyed. In some cases it is produced by pulmonary emboli, which are accompanied by hemorrhagic infarct and clogging of the blood in the bronchioles ; in others it is related to certain diseases of the arteries, to rupture of aneurisms (Hartmann), and to ulcerating pulmonary neoformations (Rossignol). Oftentimes it occurs in the course of passive pulmonary hyperemia and contagious pneumonia, and in some other infectious diseases (anthrax, septicemia).

Traumatic pulmonary hemorrhages need not be spoken of here.

Hemorrhages of the bronchial tubes and larynx are usually produced either by foreign bodies or by tubercular or glandered ulcerations. Csokor has described a case of hemorrhage of the submucous connective tissue of the bronchi.

Symptoms. A characteristic symptom of hemoptysis is a discharge, from the nostrils and mouth, of a variable but sometimes considerable quantity of clear, red frothy blood, which frequently escapes in jets. We observe at the same time an increase in the respiration and dyspnœa ; the animals undergo attacks of suffocation, cough frequently, tremble, are anxious, restless, and perspire freely. On auscultation we find large bronchial, bubbling râles. When the loss of blood is excessive the patients show the usual symptoms of profound hemorrhages, and are attacked by spells of vertigo ; the mucous membranes become pale, the skin is cold, and the pulse small or almost imperceptible.

In some subjects the hemorrhage ceases, not to appear again ; in others it returns within a few hours or a few days. Death may occur rapidly. In a large number of cases, however, the animals

recover permanently. (For a differential diagnosis of this disease and blood vomiting, see Hematemesis.)

Treatment. The treatment depends upon the cause of the hemoptysis. Bleeding is indicated when the hemorrhage is the consequence of pulmonary hyperemia or pneumonia. Cold compresses applied to the thorax are generally useful. Internally we may try styptics: ergot of rye, tannin, alum, sugar of lead, sulphate of iron, etc. Inhalations of the vapors of chloride of iron and vinegar (Viborg) have been efficient in a few cases. From a prophylactic point of view we must avoid excitement, movements, high temperature in the stables, and over-heating food.

PNEUMONIA.

GENERAL CONSIDERATIONS UPON THE DIVISIONS OF PNEUMONIA. In the lungs inflammation assumes numerous forms from the standpoint of its essential characters, its causes, location, extent, and course.

1. According to the anatomical characters of the exudate we recognize *croupous* or *fibrinous*, *catarrhal*, *hemorrhagic*, *purulent*, *necrotic*, and *desquamative pneumonia*; this consists of an over-production of the pavement epithelium of the aveoli. In this division we may also include *inflammatory œdema* if we consider it as a serious type of pulmonary phlegmasia.

2. According to the seat and extent of the inflammation we distinguish: *a. lobar*, *lobular*, and *miliary pneumonia*, according to the diffuse or circumscribed character of the process to the large or very limited centres of the pulmonary parenchyma; *b. interstitial* and *interlobular pneumonia*: the first is characterized by a migration of leucocytes in the interstitial connective tissue; it is followed by proliferation of the elements of this tissue; in the last the lesion is essentially an intense phlogosis of the interlobular lymphatic apparatus, the inflamed pulmonary parenchyma is divided into small islands by thickened lymphatic spaces (contagious pleuro-pneumonia); this form has also received the name of *dissecting pneumonia*; *c. of broncho-pneumonia* and *pleuro-pneumonia*.

3. From an etiological point of view we recognize: *a. primitive, idiopathic, infectious, glanderous, tuberculous, actinomycotic, mycotic, verminous*, and *traumatic pneumonia*; *b. pneumonias of aspiration, of inhalation, and deglutition* (traumatic pneumonia); *c. hematogenic*,

metastatic, hypostatic, pleurogenic pneumonias, etc.; *d. infectious* pneumonia; contagious pleuro-pneumonia, contagious pneumonia of the horse, pulmonary phlegmasias, which occur in the course of influenza, disease of young age, pyemia, septicemia, etc.; *e. a pneumonia of aspiration*, also called *pneumonia vagus*, which is produced by section of the pneumogastric.

4. According to their course, we divide pneumonia into *benign, malignant, abortive*, and *ephemeral* (lasting but one day) forms, also the *fixed* form (which remains limited to the region primarily affected), *migrative, erratic* (this last is an intermittent migratory pneumonia, which proceeds by fits and starts, and affects successively certain pulmonary areas); these latter forms have enabled us to establish a relation between pneumonia and erysipelas. Formerly malignant pneumonias were also qualified as "*typhus*" or "*adynamic*," because they are accompanied by extreme weakness. They are given the epithet *bilious* when they coexist with icterus produced by alterations in the liver or by duodenal catarrh. In all the animal species we find various forms of pneumonia.

The principal are: in the HORSE, *contagious pneumonia, primary catarrhal* and *croupous pneumonia*; in the DOG, *catarrhal pneumonia* of canine distemper; in the OX, *tubercular* and *interlobular pneumonias* (contagious pleuro-pneumonia); in the SHEEP, *verminous pneumonia*.

The varieties offered by pneumonia are too numerous to be studied in detail. Directing our course from a clinical and practical point of view we shall limit ourselves to the following forms:

1. *Primary croupous pneumonia* of the horse, ox, and other domestic animals;

2. *Catarrhal pneumonia*;

3. *Pneumonia which is produced by foreign bodies*;

4. *Mycotic pneumonia*;

5. *Interstitial pneumonia*;

6. *Metastatic pneumonia*.

For *contagious pneumonia* see Epizootic Diseases.

1. Croupous Pneumonia.

a. PRIMARY CROUPOUS PNEUMONIA OF THE HORSE.

Etiology. No doubt can exist at the present time on the specification of primary croupous pneumonia of the horse, but the nature

of the infectious processes which develop in the lungs is entirely unknown. Among the external causes of this disease we must particularly mention excessive exertions and cold. When either one or the other of these factors occurs in its early stages it begins evidently with active hyperemia of the lungs. It may also be determined by the inhalation of hot air and irritating vapors (smoke, vapors of chlorine, sulphurous acid). Finally it possibly may be sometimes due to a miasmatic origin.

Of late years the question has been debated whether there exists in the horse, besides contagious pneumonia (infectious pleuro-pneumonia), a croupous pneumonia which is produced by a specific morbid agent. We must answer in the affirmative, as we have already done in the first edition of this book; nevertheless, the results of Schütz's researches compel us to enlarge the clinical field of contagious pneumonia. In the present state of our etiological and bacteriological knowledge of pulmonary inflammations we must liken to this latter disease all forms of pneumonia in which we find Schütz's bacillus, whether it be acute or chronic, lobar or lobular, typic or atypic, malignant or benign, croupous or necrotic, and accompanied or not by pleurisy. But pneumonic infection may certainly be produced by several microbic agents. Rust¹ has shown that there are stables in which contagious pneumonia exists and where cases of sporadic pneumonia are observed; at the autopsy of the subjects which died from this latter Schütz's bacillus was not found. This fact establishes that apart from the micro-organisms of contagious pneumonia there are pathogenic agents whose nature is unknown, and which generate primary or idiopathic pneumonia.

Sporadic croupous pneumonia of the horse is similar to sporadic croupous pneumonia of the ox, a disease which is independent of contagious pleuro-pneumonia.

In our first edition we studied in the chapter on "primary croupous pneumonia of the horse" *epidemic lobar croupous pneumonia*, which constitutes, from both the clinical and anatomical standpoints, a morbid entity which is very different from *lobular contagious pneumonia*. We have distinctly separated it from the last by describing it as a *lobar pneumonia* of typical course, without necrosis and important participation of the pleura in the morbid process. Schütz having found in contagious pneumonia and in

¹ Rust: Statis. Veterinär.-Ber. für die Preuss. Armee, 1887.

our epidemic lobar croupous pneumonia the same pathologic bacillus, and his assertions not having been contradicted, we think it our duty at the present time to describe epidemic lobar croupous pneumonia in the chapter upon contagious pneumonia as a particular clinical and anatomical form of this latter disease. Primary pneumonias of the horse should, therefore, no longer be considered with sporadic croupous pneumonia and catarrhal pneumonia.

Pathological anatomy. Primary croupous pneumonia is characterized anatomically by inflammation of the alveoli and bronchioles and by the formation of a fibrinous exudate which coagulates very rapidly inside of the lobulo-alveolar cavities. When phlegmasia ends in resolution, the pulmonary parenchyma generally resumes its normal character. This pneumonia takes the *lobar* type, that is to say, it extends and remains confined to a more or less considerable portion of the lung; the lower lobes are ordinarily affected. In a majority of cases it is unilateral. Anatomico-pathological alterations vary with the period during which they are observed. For a long time we have recognized the following stages:

1. The period of engorgement, which lasts about twenty-four hours. It is marked by an intense congestion of the diseased portion, which is red, tumefied, œdematous, retains the imprint of the finger, and creaks less when incised; the section is smooth, shiny, moist, and sometimes hemorrhagic; the capillaries are distended and filled with blood. The alveolar and bronchial cavities are filled with a sero-albuminous liquid (inflammatory œdema of the alveoli) containing red corpuscles proceeding from hemorrhages, also leucocytes coming from the bloodvessels by a diapedesis and numerous desquamated epithelial cells; the interstitial tissue is the seat of an œdematous infiltration; in the bronchial tubes we find a frothy liquid with very fine bubbles. The air has access neither to the alveoli nor the bronchioles, not even into part of the bronchi.

2. The period of *red hepatization*, which begins with coagulation of the fibrinous exudate, and the average duration of which is two days. When the fibrinogenous substance of the blood plasma and the fibrinoplastic substance of the leucocytes are united, microscopic fibrinous threads which are arranged like a fine network are formed in the coagulated exudate. The lungs, which are much tumefied and very dense, sink in water; sometimes its consistency is similar

to that of liver or of rubber; at other times it is friable; oftentimes it is found flabby, soft, and undergoing liquefaction. Its color is usually reddish-gray; when the inflammation is very intense, circumscribed hemorrhages exist in more or less large numbers in the parenchyma, and the section shows small blackish islands which are disseminated in the hepatized tissue (granitic section); it is very rarely uniformly red; when there is any hypostasia the diseased portions are of a steel-blue color and of the consistency of the spleen (splenization). On the darkest regions the section is granular, strewn with small prominent points formed by fibrinous masses which fill the alveoli; they become adherent to their wall and do not loosen when the tissue is compressed.

3. The stage of *yellow hepatization* is marked by a decoloration of the exudate, a destruction or fatty degeneration of cellular and fibrinous elements, a beginning dissclution and absorption of these matters, and by an abundance of migratory cells. The alveolar granulations become detached on the section by compression of the tissue.

4. The period of *resolution* is characterized by the liquefaction of degenerated exudates, by their absorption and expectoration. When this pathological evolution terminates the lungs undergo an integral restitution.

In the neighborhood of the inflamed tissue the parenchyma is often œdematous and emphysematous. The pleura which covers the diseased parts looks inflamed and dull; sometimes it is studded with hemorrhages (dry pleurisy); exudate in the pleural pouches is rare. The bronchial mucous membrane is inflamed; in some instances it is coated with a croupous exudate. The lymphatic ganglions of the lungs are obstructed, œdematous, and softened; they are quite frequently marked with hemorrhages. The gastrointestinal mucous membrane has a catarrhal aspect. The liver, kidneys, and heart sometimes undergo fatty degeneration.

In cases where the evolution of pneumonia does not follow its usual course the process may determine either suppuration or gangrene (acute type), or induration and atelectasis when the disease follows a chronic course.

Symptoms. The clinical picture of primary croupous pneumonia is in all respects similar to that of *epidemic croupous pneumonia* of the horse. The first symptom is an intense fever accompanied by a prolonged chill, which is marked by great

fatigue, muscular weakness, stupefaction, and intense redness of the mucous membranes. The temperature oscillates between 40.5° and 41.5° . According to the constitution of the animals, the number of pulsations varies between 60 and 100. During the first six days the febrile reaction remains generally stationary and presents but very slight remissions; later the temperature falls, as a rule, very rapidly. The appetite is diminished, at times almost wanting, but never entirely absent; the patients are constipated, and defecation is rare. In serious cases we observe an icteric coloration of the conjunctiva, a phenomenon which is probably due to a disorganization of a large number of red corpuscles.

Respiration is accelerated and difficult in proportion to the intensity of the fever and the extent to which that inflammation has invaded the lungs; it is especially costal; we may count from 20 to 60 inspirations per minute; the nostrils are much dilated; the expired air is warmer than usual. We hear a superficial cough, which is short and painful; this symptom is, however, sometimes wanting. There is frequently a reddish or yellowish discharge (*rusty discharge*). The animals remain standing constantly, or they lie down for a short time only; they are ordinarily found standing, anxious, immobile, with the forelegs spread; when they take a recumbent position they always lie upon the diseased side.

In the beginning and during the height of the attack percussion of the thorax gives a *tympanitic sound*, which is rarely pure; it is rather dull, and on auscultation we perceive a *moist crepitant râle* in the region of the inflamed zone. During the period of hepatization the percussion sound is *dull* and the sensation of resistance under the pleximetric hammer is much increased; on the limits of the dull region we find a tympanitic resonance; on auscultation we hear bronchial wheezing (*tubular wheezing*) during expiration. At the period of dissolution the sound of percussion again becomes *tympanitic* and the ear perceives *moist râles*.

On auscultation of the healthy regions of the lungs we hear a vesicular murmur which is stronger and harder (supplementary murmur).

In cases where the disease miscarries the percussion sounds are wanting in distinctness, and the information which is obtained by this method of exploration is of little or no value.

In a large number of patients the urine is alkaline for quite a long time—sometimes during the whole continuous period of the

disease ; but it generally becomes acid within a certain number of days. In the last stage of pneumonia it is frequently albuminous ; its specific gravity is increased ; it contains a less proportion of chlorides (pulmonary exudates) and a greater quantity of urea than normal urine.

Course and prognosis. The ordinary course of croupous pneumonia is typical. Engorgement, hepatization, and resolution follow one another in regularly determined periods. The disease reaches its height toward the fifth or sixth day ; then follow degeneration, liquefaction, and absorption of the exudates. Dulness disappears sometimes in a very short time (in twenty-four hours), but usually this phenomenon is produced in a slow and gradual way ; we recognize large bubbling râles and crepitant râles, the cough is less painful, there is a slight muco-purulent discharge, the appetite reappears, and recovery occurs in eight to fifteen days.

In other cases, which are quite frequent, the process does not go through all its phases ; it ceases at the stage of engorgement or beginning of hepatization. This abortive form comprises pneumonia, which lasts but two or three days (ephemeral pneumonia).

Death by asphyxia occurs in serious cases (typhic), which are accompanied by an exaggerated globular destruction in those where the inflammation is much extended and heart weakness extreme, or also when a generalized pulmonary œdema appears as a complication of the disease.

Pneumonia may pass into a *chronic state* in weak animals, when very old or very young, in animals which have been previously affected by bronchitis or bronchiectasis. A connective-tissue neoformation is sometimes produced which leads to pulmonary induration, which is especially indicated by persistence of the dulness and dyspnoea (see Interstitial Pneumonia). At other times phlegmasia produces *suppuration* or a *limited mortification* ; the fever is intense, there are numerous chills, the discharge is abundant, the expired air is fetid, etc., and we observe the ordinary symptoms of septic infection. In certain cases this chronic form is cured within from six to eight weeks ; in others it leads to "chronic pulmonary phthisis" (wind-broken) ; finally, in others it determines death. This is generally occasioned by septic infection or by a secondary myocarditis.

But these complications are very rare, and the prognosis of

croupous pneumonia is very favorable in the majority of cases. Out of 100 patients only a few die.

Differential diagnosis. Primary croupous pneumonia is distinguished from *catarrhal pneumonia* by different characters. Its initial stage consists of an active pulmonary hyperemia, while catarrhal pneumonia begins with bronchitis. This latter form of pulmonary phlegmasia has an irregular course; it ends frequently by atelectasis and induration; it is *lobular*; on percussion we recognize numerous limited zones of dulness corresponding to the inflamed pulmonary islands.

We differentiate pneumonia from *pleurisy* by taking as a base the typical course of the former and the symptoms which are elicited by a physical examination of the lungs. In pneumonia the zone of dulness is limited by an irregular line which ascends and descends successively, the bruit of percussion is sonorous and the heart action is strong. In pleurisy the line of dulness is horizontal and formed by the level of the exudate; the percussion bruit is dull, the precordial impulse weaker than in a normal state (the heart is thrown back by the pleuritic liquid), and the respiratory bruits are generally wanting (except in certain cases of pneumopleurisy). A recognition of friction bruits enables us to affirm the existence of pleurisy.

Primary croupous pneumonia is distinguished from *contagious pneumonia* by its essentially sporadic character. Since the appearance of Schütz's works we know that this latter is produced by specific agents, but a bacteriological examination is of secondary importance from a differential diagnostic standpoint; first, it is almost impossible in the living animal, and, among the numerous pulmonary microbes it is often very difficult to recognize the pathogenic bacilli.

Treatment. Croupous pneumonia is a disease with a definite and generally benign course, and does not require very active treatment. Dietetic care constitutes the principal indication.¹ Cold compresses applied upon the thorax and removed every ten minutes are generally advantageous.

In cases where the disease deviates from its normal course the therapeutic intervention varies according to the cases.

When hyperthermia is intense and the animals weak we must

¹ Horses which are seriously affected should be sustained on milk (five to fifteen litres daily); most animals drink it with avidity.—N. D. T.

use antifebriles. Quinine and alcohol have but little value; anti-pyrine, antifebrine, phenacetine, in doses of 20 to 25 grammes, are much more active. If we recognize the symptoms of cardiac exhaustion, we must make use of digitalis; we give this latter in a dose of 8 to 10 grammes (powdered leaves) in the form of an electuary, associating it with alkalines.

When resorption operates slowly we must resort to inhalations of steam and to Priessnitz's compresses applied on the thorax; internally we administer alkalines, especially solution of acetate of potash (100–150 grammes) in an electuary with powdered juniper berries. In gangrene of the lungs we must direct inhalations of tar or cresol. Dry frictions, cold rectal injections, and the application to the pectoral walls of a sinapism prepared with essence of mustard (6–8 per cent.) are also useful remedies. The sinapism acts at the same time as an antipyretic and derivative. In old weak animals alcohol renders good service as a moderator of the nutrition; it is given in a dose of 100 to 200 grammes in drinking water. Wine, in a dose of one-half to one litre per day, is still more useful.

b. NON-CONTAGIOUS CROUPOUS PNEUMONIA OF THE OX.

REMARKS ON THE RELATIONS OF NON-CONTAGIOUS CROUPOUS PNEUMONIA, WITH CONTAGIOUS PLEURO-PNEUMONIA OF THE OX. Gerlach, Haubner, and others have for a long time denied the existence, in the ox, of pulmonary phlegmasia other than contagious pleuro-pneumonia. Gerlach, in his *Treatise on Legal Veterinary Medicine* (1872), classes with contagious pleuro-pneumonia all cases of marbled pulmonary hepatization. At the present day the fact can no longer be contested that we find a non-contagious primary pneumonia in bovines. Without doubt, among the numerous cases which have been mentioned, a certain number are related to pneumonias produced by liquids which have fallen into the trachea, also by foreign bodies coming from the plexus, by worms, etc.; and in some cases it is in fact contagious pleuro-pneumonia which, for want of material of contamination, runs its course like a sporadic disease. But, from abstracts made of these particular affections, there are always quite a large number of well-detailed cases which preclude the existence of sporadic pneumonia.

The causes of this form of pulmonary phlegmasia are little or not at all known. It is possible that they consist of physical or chemical irritating substances which reach the lungs with the in-

spired air; it is also possible that this disease is of a miasmatic nature (?), infectious and non-contagious, exactly as in the horse. New researches are necessary in order to dissipate the obscurity which also covers its etiology.

Pathological anatomy. This pneumonia is characterized by the same essential alterations as in that of the horse. The differences which are recognized are due to the particular structure of the lungs of the ox; in this species the interlobular lymphatic spaces are the seat of an inflammatory process more or less intense, and which they transmit rapidly to the pleura. The pulmonary inflammation takes in preference a lobar type, the four periods—*engorgement*, *red hepatization*, *yellow hepatization*, and *resolution* are distinctly marked. In the stage of hepatization the color of the section is dark red or blackish-brown. Secondly, the interlobular connective tissue becomes the seat of an œdematous infiltration or of a slight fibrinous exudation, the product of which is deficient in white corpuscles. The pleura is more seriously affected than in the horse; sometimes we find dry pleurisy, at other times and mostly a serous or sero-fibrinous pleurisy.

Instead of following the example of other authors in giving a general description of the lesions of this trouble, it has seemed to us preferable to mention summarily the anatomo-pathological examination which we made in one case.

In the month of September, 1885, a newly-bought cow was affected by pneumonia, to which disease it succumbed within four days. At the autopsy absolutely nothing abnormal was found in the abdominal organs; there was no trace of perforation of the first gastric compartments by a foreign body. Two-thirds of both pleural pouches were filled by a serous reddish-yellow exudate. We observed fibrinous clots upon the pulmonary and diaphragmatic folds; the former was covered by it on the hepatized regions; upon the costal pleura the exudate was diffuse and uniform. The pericardium particularly was much coated by it, and it was spread over a wide surface of the costal walls (sero-fibrinous pleurisy); its internal fascia was shiny, and its bloodvessels appeared much injected; the connective tissue of the neighborhood was the seat of a gelatinous infiltration; the epicardium, endocardium, and large bloodvessels were infiltrated by the coloring-matter of the blood; the heart cavities contained a soft blood-clot of a dark color.

The right lung did not show any important alteration; the lower

portions (especially the anterior lobules) were almost deprived of air and filled with liquid (slightly atelectatic). The anterior lobe of the left lung offered the same alterations (dark red, hard, heavy, hyperemic, and poor in air). On the anterior extremity of the principal lobe a mass of the size of two fists was observed, which did not sink and showed a blackish-red color through the tumefied dull pleura, which was covered by a fibrinous exudate. This portion of the lung had the consistency of rubber. The sections made into its depth presented a blackish-red color (red hepatization). The phlegmasic process was distinctly limited on the periphery; in the neighboring lobules and in their interstices we observed an œdematous inflammatory infiltration.

Hepatization was regularly divided in the altered portion, the tissue of which resembled that of the liver (the granite-like aspect which is remarked in the horse did not exist). *The alterations were similar in the several lobules of the affected region*; they presented the same characters and the same age; everywhere the same red hepatization was found, also a coloration and uniform consistency of the exudate. The interstitial connective tissue was slightly tumefied and infiltrated with a yellowish or grayish-yellow gelatinous liquid according to the location; the length of these fibres varied from 1 to 2 millimetres.

All the bloodvessels of the diseased part were obliterated by recent thrombi, and the bronchial tubes were partially filled with a frothy liquid and blood-clots; the mucous membrane of these canals was tumefied, of a diffuse red color, and strewn with hemorrhages.

Notwithstanding that this animal had remained for several days in a stable containing thirty-six cows, and that it died there, two months later none of these animals had been affected by pneumonia.

This simple clinical fact seems to us to be demonstrative of the existence in the ox of a croupous pneumonia which is independent of contagious pleuro-pneumonia.

Symptoms. The symptoms are nearly the same as in the horse: high fever (from infection), serious general symptoms, dyspnœa, complaints, cough, at first a normal percussion sound, later becoming tympanitic and dull; on auscultation, in the beginning, crepitant râles, then bronchial wheezing, and finally large bubbling râles. Frequently croupous pneumonia of the ox has a rapid course

and a fatal termination. When it ends in death, this occurs during the period of hepatization, toward the third or fourth day.

Differential diagnosis. It is very difficult to differentiate simple croupous pneumonia from *peripneumonia*. The following are the most important points on which we may depend:

1. Croupous pneumonia is always observed in a sporadic state and mostly in stables where contagious peripneumonia has never been recognized.

2. It is quite distinctly characterized by its acute or subacute evolution and by a regular succession of its various periods: *engorgement, red hepatization, yellow hepatization, and resorption*. In its initial stage, when the fever is very high, we do not recognize any pulmonary dulness. Peripneumonia has an irregular atypical course, and percussion shows dulness from the start.

3. The anatomical alterations in these two affections differ considerably. In croupous pneumonia all hepatic centres show alterations of age, color, and identical consistency: in certain cases the interstitial lymphatic system (interlobular lymphatic spaces) does not participate at all in the morbid process; in others it is the seat of a slight oedematous infiltration; in some very serious cases it contains a slight fibrinous exudate in which few leucocytes are present. In peripneumonia the differences of age and aspect of the different hepatized centres are characteristic. The participation of the lymphatic interlobular apparatus in the process is also a constant lesion. We find always an intense inflammation of the interstitial lymphatic bloodvessels (lymphangitis) with thrombosis of these vessels and great inflammatory infiltration of the connective tissue which afterward becomes hypertrophied. Finally, there exists a fibrino-exudative pleurisy, a consequence of the extension to the serous membrane of pulmonary phlegmasia.

Authors have attached great importance to *marbled hepatization* as a diagnostic symptom. For Gerlach this lesion is proper to peri-pneumonia; for the majority, and among these Bruckmüller, it would be but an alteration common to the many varieties of pneumonia.

The whole question consists in having an understanding concerning the value of the expression "marbled hepatization."

If we consider as such the modifications of the pulmonary tissue which are marked by yellowish or gray-yellowish trails (infiltrated interlobular connective tissue), and by small red islands (hepatized

pulmonary lobules), this alteration can be recognized in several varieties of pneumonia and in the different species (traumatic pneumonia); for it is not unlikely that the interstitial connective tissue may be much infiltrated in other animals than the ox and pig.

But if, with Gerlach, we consider marbled hepatization as essentially characterized by *pulmonary lobules in different periods of hepatization*, and separated by thick irregularities of interstitial connective tissue which are infiltrated by a yellowish exudate, we are forced to admit that marbled hepatization is a lesion which is pathognomonic of contagious pleuro-pneumonia; this anatomo-pathological variety is, in fact, never observed in non-contagious acute croupous pneumonia.

Marbled hepatization is always characterized by the existence of small lobular multilocular territories (brownish, yellowish, grayish, etc.), which are separated by thickened interstitial lymphatic walls.

Treatment. Croupous pneumonia of the ox has a very rapid course. We rarely have the opportunity to exercise the means which are required for its treatment. This is, besides, identical with that of the horse. Its principal agents are cold compresses on the thorax, cold infusions or rectal irrigations and antipyretics. Among these latter alcohol is particularly advisable.

c. CROUPOUS PNEUMONIA OF OTHER SPECIES.

Croupous pneumonia of other domestic animals is little known. Special literature rarely contains observations of it, and the descriptions which are given in most of the treatises on pathology are purely theoretical. This void has yet to be filled.

1. According to Röhl, croupous pneumonia is frequent in the DOG, and ordinarily reaches the posterior lobes of the lungs. We cannot confirm these assertions. According to our observations, lobar croupous pneumonia is very rare in the dog, and its symptoms and alterations are nearly the same as in the horse. The catarrhal form is the most common in the canine species.

2. In the CAT we have seen croupous pneumonia more frequently than in the dog.

3. In the PIG this pneumonia is quite common in animals which are driven to distant fields during seasons of great heat or intense cold. Nevertheless it is relatively very rare if compared with infectious pneumonia (Schweineseuche). Up to the present time no exact description has been given of it.

4. In the SHEEP Roloff has especially observed acute croupous pneumonia in lambs from two to three weeks old. In almost all cases the inflammation was confined to the anterior lobes of both lungs or of one only. The animals generally die during this condition as a consequence of œdema of the non-hepatized pulmonary regions. Cox has reported similar observations.

2. Catarrhal Pneumonia: Broncho-pneumonia.

Etiology. Catarrhal pneumonia is usually excited by causes similar to those which set up acute bronchitis. Besides cold they include irritating agents which are introduced into the lungs by inspired air (this is contrary to that which takes place in hematogenic croupous pneumonia): dust, sand, and other foreign bodies, fungi, and infectious matters. This special pathology explains the enzootic character of the disease in young animals (calves, lambs, young pigs). It is often developed at the expense of acute bronchitis, either by a propagation to the pulmonary alveoli and interlobular tissue of the phlegmasia of the bronchioles, or by aspiration into the alveoli of bronchial secretions loaded with infectious elements.

In the majority of cases broncho-pneumonia occurs as an epiphenomenon of various infectious diseases, and affects especially young or debilitated animals. In the dog, where it is very common during the course of canine distemper, it represents the ordinary form of pneumonia. In the ox it is also quite frequent and of great importance, because of the likelihood of an infection with tubercular bacilli. It is more rare in the horse. In the sheep, pig, calf, and gallinaceous species it is found in the form of verminous pneumonia.

Hypostatic pneumonia is another variety of catarrhal pneumonia. It is produced when the return of the blood contained in the pulmonary bloodvessels is hindered by an increasing weakness of the heart; its initial stage is therefore a passive pulmonary hyperemia. It is localized on the lower portions of the lobes, where the blood accumulates through the action of gravity. It is seen in the course of tetanus, of chronic affections of the heart, of blood diseases, etc., especially in weak animals, and particularly in the sheep.

Pathological anatomy. Catarrhal pneumonia is characterized anatomically by its distribution in centres; it is *lobular*. At times there is but one small island; in other cases the lesion unites sev-

eral confluent islands; sometimes the disease is *disseminated*, and there is a large quantity of small miliary inflammatory nuclei (distemper in the dog).

This form of pneumonia starts also by hyperemia, hemorrhage, exudation, a leucocytic infiltration which is more or less marked, and an abundant epithelial desquamation; there is no fibrinous exudate. According to the absence or predominance of one or another of the inflammatory products, section of the hepatized lungs shows a black, dark red, or brown coloration; later, when the cellular elements increase, the coloration becomes successively red, gray, yellowish-gray, or yellowish-white. On the surface of the section the broncho-pneumonic centres are smoother than in croupous lobar pneumonia; it is finely granular; the tissue of these centres, which is hard and deprived of air, makes a slight projection on the cut surface. Compression of the section in the early stages of the disease causes an oozing from the alveoli and bronchi of bloody or dark chocolate-colored liquid, which in the later stages becomes reddish or pale gray. The healthy pulmonary tissue of the neighborhood is the seat of a collateral hyperemia.

When broncho-pneumonia is disseminated the surface of the lungs is studded with small grayish-white purulent centres, which vary in size between that of a poppy- and a hempseed. Sometimes we observe a diffuse purulent infiltration of large portions of pulmonary tissue.

The most important of the consecutive alterations is *atelectasis*. It is developed by obstruction of the bronchioles (obstruction of atelectasis) or by accumulation of the exudate in the bronchioles and the alveoli. On points which no longer contain air we observe passive hemorrhages, agglutination and a union of alveolar walls, and a connective-tissue neoformation and thickening of the walls (collapse and pulmonary induration). Atelectatic regions are recognized by their dark red-blue coloration; sometimes they project from the surface of the lungs. The hemorrhages cause a slaty pigmentation (slaty pulmonary induration). Around and in the neighborhood of the broncho-pneumonic centres the pulmonary tissue is frequently emphysematous; on their surface the bronchi become dilated, the mucous membrane of these ducts is inflamed, and ectasies are developed on their surface, the pleura is thickened and rough. The bronchial ganglions are tumefied, œdematous, and sometimes infiltrated with pus.

Among other lesions seen in broncho-pneumonia let us also mention suppuration and gangrene of pulmonary tissue. Foreign bodies which have reached the lungs by accident may become surrounded by a connective-tissue shell of varying thickness (nodular induration).

Symptoms. In its beginning catarrhal pneumonia is marked by symptoms of acute bronchitis, to which it is consecutive in a large number of cases. The general temperature rises; respiration becomes accelerated; the cough is short, painful, often abortive, especially in the dog. When the broncho-pneumonic centres, which are often of quite considerable size, are situated superficially, percussion enables us to recognize dull circumscribed zones which are usually located toward the lower portions of the lungs, and on the circumference of which the resonance is tympanitic. Bronchial wheezing (tubal wheezing) is rare; respiratory bruits, which are weakened or have completely disappeared, are replaced by râles.

The course of the disease is sometimes very rapid; however, generally it is slow and chronic, and prolonged for weeks or months. In some cases recovery takes place within two or three weeks; the inflammatory products are absorbed or expectorated, râles become more and more moist, fever diminishes, respiration gradually returns to its normal rhythm, and the cough disappears; under other circumstances complications are produced, among which pulmonary induration (broken-wind) and "pulmonary phthisis" are the most important.

Suppuration and necrosis are rare; they are indicated by a very high fever, considerable emaciation, and great weakness; the cough ceases, and collapse is more and more profound. Death may be determined very rapidly, either by an extension of the morbid process to a great part of the lungs or by a secondary pulmonary oedema.

The prognosis is especially serious in young and weak animals.

Differential diagnosis. Catarrhal pneumonia differs from croupous pneumonia by its slow, chronic, atypic course; it is often consecutive to bronchitis; percussion reveals the existence of dull circumscribed zones; the rusty discharge is wanting; finally, its progress is that of a serious disease. In the horse it is the rarest form of pneumonia; in the dog it is the most frequent.

A distinction from capillary bronchitis (bronchiolitis) is sometimes difficult. Hyperemia, a painful cough, lobular dulness, and the absence of vesicular murmur are important diagnostic symp-

toms; but it is very rare to find these united; very frequently we do not observe any dulness on account of the deep location of the hepatized centres (distemper of dogs). It is almost impossible to recognize positively when bronchiolitis is transformed into broncho-pneumonia.

Treatment. The treatment of catarrhal pneumonia comprises the same indications as that of acute or chronic bronchitis. Nevertheless the antipyretics and tonics for the heart (alcohol, digitalis, and caffenin) are more specially recommended. When the disease exists in a chronic state we must combat it with dissolvants and expectorants (alkalines, apomorphia, essences, and resin), cresol inhalations and water compresses. This treatment should be aided by hygienic care and good feeding.

3. Traumatic Pneumonia: Pulmonary Gangrene.

Etiology. In its beginning pneumonia produced by foreign bodies consists of a catarrhal phlegmasia, lobular or in centres. In veterinary medicine it is usual to devote a special chapter to it.

We generally recognize two varieties: 1. *True pneumonia which is produced by foreign bodies*; 2. *Traumatic pneumonia*.

1. *Pneumonia which is produced by foreign bodies*, in the restricted sense of the word, is the result of the action of irritating substances which reach the lungs by way of the trachea and bronchi. In its first stage it represents a lobular catarrhal pneumonia; soon the inflammatory process becomes intense, especially in the ox, and produces a croupous exudate and pulmonary gangrene. We ought to classify in this group pneumonia which is produced by the inhalation of dust, verminous and mycotic pneumonia (moulds); but in practice we restrict the name pneumonia by foreign bodies to the disease of the lungs produced by food or liquids which have *mis-carried*.

a. Alimentary pneumonia is especially observed in the horse; more rarely in the ox. In solipeds the diseases of the pharynx, of the encephalon, and tetanus occasion this miscarriage quite frequently, that is to say, the entrance of alimentary matter into the trachea. In the cow its most frequent cause is the paralytic form of parturient fever. Aphthous pharyngitis (Lydtin), retro-pharyngeal abscesses which open spontaneously, putrid bronchial secretions which become gradually infiltrated into the most dependent portions of the lungs, and coughing spells during rumination

(Eisenblätter) may also produce it. Feser has observed it after the administration of large doses of physostigmine. In all species we have recognized in the foetus pneumonias due to penetration (by inspiration) into the lungs of amniotic liquids and intestinal mucus. In the majority of cases it might be to these causes that pneumonias of the newborn ought to be ascribed.

Let us also mention *experimental vagus pneumonia*, which is determined by paralysis of the laryngeal nerves, a lesion which permits the penetration of food into the larynx and trachea.

b. Medicamentous pneumonia (pneumonia medicamentaria) is of more importance than alimentary pneumonia. It is observed in the horse, ox, and pig. It is determined by a defective administration of drinks. Their introduction into the nasal cavities (much practised by empirics), tractions which are performed on the tongue during that operation, occlusion of the nostrils, compressions upon the laryngeal region, a too great extension of the head, administration of an excessive quantity of medicinal liquids, mixtures which deposit sediments and which require shaking before being used, vegetable powders, infusions of aromatic flowers, irritating, bitter, viscous matter; such are its usual causes. In the pig we must take into account besides a resistance which the animals offer to the administration of therapeutic liquids; this animal utters groans and the liquids drop into the gaping larynx. In the horse a too high position of the head is particularly dangerous when the animals are attacked by coughing spells during the swallowing of drinks. Formerly, when death occurred on account of *miscarried food*, it was explained by a claim of poisoning, even when entirely harmless medicines were concerned, such as linseed oil, pepper, etc. (Hertwig).

Intra-tracheal injections, which are used at the present time, have also sometimes set up gangrenous broncho-pneumonia.

The degree of intensity and modes of reaction of the bronchial mucous membrane and of the pulmonary parenchyma depend upon the nature of the foreign body. Distilled water, fountain water, and various non-irritating medicinal solutions may be absorbed without leaving after them any troublesome consequences. Insoluble medicines, irritants, or caustics produce phlegmasias, the acuteness of which varies from a simple catarrh to a diphtheric, necrotic, or gangrenous inflammation.

2. In *traumatic pneumonia*, which belongs to the domain of sur-

gery, the foreign body penetrates into the lung through the surface of the organ. It is quite frequently observed in the ox during the course of traumatic pericarditis. In the dog it is sometimes determined by needles which penetrate into one of the lobes in traversing the œsophageal shields; in this form of the disease the symptoms of pleurisy ordinarily predominate. We must also mention pneumonia by contusion (Schütz), which complicates traumatism, acting upon the thorax (falls, injuries, external violence), and wounds of the lungs which are consecutive to fracture of the ribs or to penetrating wounds of the chest.

Pneumonia by foreign bodies is one of the most frequent causes of pulmonary gangrene. But in the horse this may also be produced during the course of contagious pneumonia (necrotic pneumonia), and of serious croupous pneumonia with arrest of the circulation in a large portion of the lungs after hemorrhagic infarctus and embolic processes (see Metastatic Pneumonia); finally, sometimes it is occasioned by decomposed bronchial secretions; it occurs then as a complication of bronchiectasis.

Pathological anatomy. Pneumonia by foreign bodies is a complex morbid process formed by an association of catarrhal, croupous, gangrenous, and necrotic phlegmasias.

Pulmonary gangrene is characterized by an ensemble of necrotic and gangrenous lesions, which are produced by a putrid, bacterial pneumonia. In the beginning the inflammation is localized on the bronchi, then by degrees it extends to the peribronchial connective tissue and to the pulmonary parenchyma. This latter shows successively the symptoms of catarrhal, croupous (especially in the ox), and diphtheric inflammation. The diseased pulmonary territories, which protrude more or less on the surface of the cut, are at first blackish-red, then reddish-gray, and later yellowish; their resistance increases; the air has no longer access therein. We very rarely succeed in discovering foreign bodies in the centre of the lobules. By degrees the affected regions undergo infiltration and purulent wasting. In certain cases numerous abscesses are developed, which soon become confluent and transformed into gangrenous centres, when they communicate with a bronchial tube. *Medicamentous pneumonias*, which are generally bilateral, are localized on the base of the lungs or on the surface of the large bronchial tubes.

The gangrenous pulmonary tissue forms a viscous, blackish, or

grayish mass, similar to amadou, which possesses no consistency, becomes separated, liquefied, and reduced to fetid ichor, of bad aspect, and contains the products of decomposition of the albumin, fat (leucine, tyrosine, fat acids, ammonia, sulphuretted hydrogen), and a considerable quantity of figured elements (agents of putrefaction, remains of parenchyma, pus, corpuscles, various detritus, crystals of tribasic phosphate, etc.). According to Filehne, we also find in it a ferment similar to trypsin, which dissolves the elastic tissue of the lungs.

Around these gangrenous centres the pulmonary parenchyma is hyperemic and œdematous. In the horse we sometimes meet with a characteristic tumefaction of the interlobular connective tissue, which forms large gelatinous bands similar to those of peripneumonia of the ox. On the pleura we frequently recognize lesions of a purulent or hemorrhagic and putrid inflammation; the pneumothorax and pyopneumothorax are not rare. In cases where gangrene is older a disjoining furrow is formed around the necrotic centre. The mucous membrane of the bronchi and of the trachea is at times infiltrated with pus or ichor. The healthy pulmonary tissue is often emphysematous; we have several times observed subpleural, mediastinal, and even subcutaneous emphysema.

In other places we find localized septic infection and pyemia, black blood of a tar-like aspect, hemorrhages, tumefaction of the different viscera, metastasis, etc.

Symptoms. In the beginning the manifestations of pneumonia by foreign bodies are those of lobular catarrhal pneumonia; they often pass unobserved, but on exploration of the thorax we recognize significant phenomena on the surface of the points where the gangrenous centres and cavities exist.

Its principal symptoms are :

1. A disagreeable, and later a very fetid, odor of the expired air, which is perceptible from both nostrils; it is often accompanied by a bad-looking discharge, which is infectious and loaded with pieces of mortified pulmonary tissue. In some cases of advanced gangrene this fetid condition of the air and discharge may be wanting.

2. Modifications which occur in the resonance and normal bruits of the lungs. When cavities are formed, and if they are not located too deeply, percussion gives a tympanitic sound like a cracked pot, or sometimes it is metallic. On auscultation we per-

œive râles, bronchial and amphoric wheezing, and gurgling bruits (glou glou).

3. A fever, ordinarily intense. It is accompanied by chills, also by a great acceleration of circulation, with weakening of the pulse (in the horse we count from 80 to 120 pulsations per minute), depression, drowsiness, stupefaction, profuse diarrhea, and symptoms which distinctly characterize septic fever. Our own experiences have demonstrated that in some cases we may observe the preservation of appetite and a general apyretic condition, notwithstanding the existence of cavities of large extent.

Diagnosis. According to the preceding considerations, the diagnosis of pulmonary gangrene is based on the fetid odor of the expired air, on the existence of cavities, and the presence, on a microscopic examination of the discharge, of elastic fibres or other débris of the pulmonary parenchyma; the latter evidence assures a diagnosis in all cases. If this is difficult to establish in the beginning, and as long as the causes of the disease are obscure or unknown, we may adopt it as a rule that acute pulmonary diseases appearing during the course of or after colics, pharyngitis, encephalitis, parturient fever, tetanus, anasarca, diseases of the œsophagus, etc., must be considered *à priori* as pneumonias produced by foreign bodies

Differential diagnosis. This pneumonia and pulmonary gangrene may be confounded with contagious pleuro-pneumonia, bronchiectasis, pulmonary phthisis, diseases of the teeth, of the bones of the head, and of the maxillary sinus. These latter diseases have as manifestations common to the pulmonary processes concerned only fetidity of the expired air, and this symptom is generally observed in only one nostril.

It is sometimes difficult to differentiate between pulmonary cavities and bronchiectasis; nevertheless, in this latter disease the discharge does not contain any elastic fibres, and the symptoms of septic infection are not so pronounced. Pneumonia by foreign bodies is distinguished from pleuro-pneumonia by its acute course, by phenomena which mark pulmonary gangrene, and at the autopsy by the absence of marbled hepatization.

Prognosis. The prognosis of pneumonia by foreign bodies is serious. The greater number of patients die. Death occurs, as a rule, within a few days, sometimes only after weeks. Instances of cure are, however, not very rare even when the odor of gangrene

has already appeared; in these cases a gangrenous pulmonary centre is eliminated by suppuration (sequestered), and is rejected with the expectorated matter. Cases of this kind are particularly observed in the ox. Diffuse pulmonary gangrene is fatal.

Treatment. This is confounded with that of catarrhal pneumonia. But as soon as gangrenous lesions exist we must resort to other means. We may try to arrest the process with inhalations of cresol, tar, phenic acid, and essence of turpentine; we may also administer these remedies internally. Septic fever must be combated besides with antipyretics, especially with camphor and alcohol.

We should avoid *medicamentous pneumonia* by taking the necessary precautions in order to prevent a miscarriage in swallowing liquids, and by never prescribing these last for the pig.

4. Mycotic Pneumonia of Mammifers: *Aspergillus* Pneumomycosis.

Etiology. Pneumonia produced by moulds is commonly called *mycotic pneumonia*. In mammals (horse, ox) we find especially the *Aspergillus fumigatus* (Böllinger has described a case of bothriomycosis of the lungs). In birds (chicken, pigeon, goose, duck, swan, pheasant, flamingo, etc., parrot and other house birds) we may observe *Aspergillus glaucus*, *A. nigrecens*, and *A. fumigatus*, as well as the *Mucor racemosus* (more rarely the *M. conoïdeus*). These fungi are introduced into the organism with food or air which is loaded with moulds; they develop with ease in weak and delicate animals, and in those affected by catarrhal diseases. Mycotic pneumonia often exists in an epizootic state in poultry. In a stable where the horses had eaten mouldy hacked hay Pech observed it in seven subjects. (Concerning mycotic pneumonia of poultry, see the Addenda.)

The pathologic action of the fungi of moulds is established by numerous observations. They start from the bronchial mucous membrane, the bronchioles and alveoli, and determine by a complex mechanism—by mechanical and chemical irritation and by consumption of the pulmonary tissue—intense morbid processes, which are closely similar to pneumonia produced by foreign bodies. But these agents may be observed by accident in bronchial ectasies, in pulmonary cavities, and in lesions of pneumonia by foreign bodies (Siedamgrotzky).

Pathological anatomy. In mammals *aspergillus pneumonia*

ordinarily assumes the characters of purulent follicular pneumonia. The follicles vary in size from a hempseed to that of a pea. They may exist in large numbers, and are sometimes disseminated throughout the entire pulmonary substance; at other times they are confluent. In some cases they are formed by a connective-tissue capsule, which is filled with pus containing fungi; in others they represent very small lobular inflammatory nuclei, the centre of which is filled with large mycotic vegetations, while the periphery is separated from the healthy pulmonary tissue by a hepatized, hemorrhagic zone. More rarely we find a diffuse pneumonia, which is characterized by hepatization and inflammatory infiltration of the interlobular connective tissue (Röckl), and which presents at times a striking anatomical similarity to contagious pleuro-pneumonia. Microscopic examination enables us to establish a diagnosis. In the horse follicular pneumomycosis may simulate glanders; on the bronchial mucous membrane there sometimes exist mycotic ulcerations and proliferations; upon the surface of the diseased regions we remark dry pleuritis. In one case Martin has recognized pulmonary and hepatic abscesses which contained fungi. When the disease has evolved very rapidly its lesions are those of traumatic pneumonia with consecutive pulmonary gangrene (Pech's observation in a horse).

Symptoms. The manifestations of pneumomycosis are similar to those of catarrhal pneumonia. It is rare that percussion reveals an extended dullness; however, in a case of subacute pneumomycosis, related by Pech, a dullness which involved the inferior half of the thorax was recognized. Generally the disease pursues a chronic course; it leads to "pulmonary phthisis," which is marked by persistent dyspnoea and progressive emaciation. The discharge sometimes contains moulds, which is a very important fact from a diagnostic standpoint (Pech). In subacute cases we have observed all the clinical appearances of pneumonia by foreign bodies.

Treatment. In the majority of cases a diagnosis is only made after death. Even if established during life, it would be illusory to hope to destroy the fungi which have penetrated the lungs, or to obtain their elimination. We might, however, try inhalations of cresol, tar, phenic acid, and essence of turpentine. In large animals we may resort to intra-tracheal injections of solutions of cresol, phenic acid, salicylic acid, or sublimate. But at the present time aspergillus pneumomycosis offers only anatomical interest.

5. Chronic or Interstitial Pneumonia: Pulmonary Phthisis.

Etiology. *Interstitial* or *chronic pneumonia* is a symptomatic morbid condition related to chronic inflammatory processes of the pulmonary parenchyma and the bronchi. Among these latter we may mention first: catarrhal pneumonia, pneumonia by foreign bodies, chronic bronchial catarrh, and the ectasies which so frequently accompany it. Interstitial pneumonia is also quite often determined by tuberculous, glandered, actinomycotic pneumonia, and by contagious pleuro-pneumonia and the disease of young age. In the horse it is rare to see it set in after croupous pneumonia. We have devoted special study to the affection in order to give a typical picture of it.

Pathological anatomy. Anatomically, interstitial pneumonia is characterized by diffuse or circumscribed alterations of the peribronchial or interlobular connective tissue. The pulmonary areas invaded are very hard and dry, and creak under the knife. Their volume is diminished, their coloration is whitish or grayish-white. The newly-formed tissue is developed at the expense of the leucocytes which have emigrated into the interlobular peribronchial and perivascular connective tissue; in the beginning it represents a kind of gelatiniform infiltration, which compresses the alveoli and bronchioles, the walls of which become adherent; its mass gradually diminishes in volume or becomes indurated (connective induration or sclerosis of the lungs).

After these modifications of the pulmonary parenchyma, the bronchi and alveoli, which have remained healthy, become dilated (brouchiectasis, emphysema).

Symptoms. The symptoms of interstitial pneumonia are the expression of the decrease of the pulmonary respiratory surface; they consist essentially in dyspnœa which is more or less intense (broken-wind of the horse). In almost all cases there is a weak, short, superficial cough, which is occasioned by the chronic bronchial catarrh. A physical examination of the lungs does not reveal any important phenomenon in the rather frequent cases where the pneumonic centres are disseminated, or very small, or deeply located. In others we observe manifestations of catarrhal pneumonia. When interstitial pneumonia exists for a long time we see emaciation, weakness, dropsies, albuminuria, etc., which, with dyspnœa, constitute the train of symptoms of "pulmonary phthisis." Death occurs by exhaustion.

The treatment of interstitial pneumonia is of no avail.

In a general way we designate under the name *pulmonary phthisis* all destructive chronic pulmonary alterations which are accompanied by considerable emaciation and great weakness. We must not, therefore, consider pulmonary tuberculosis as the only disease which is capable of determining it. If in the ox (and in man) this coincidence is the rule, pulmonary glanders, catarrhal pneumonia, or that which is produced by foreign bodies, actinomycosis, tumors of the lungs, etc., may also lead to pulmonary phthisis in animals.

Among *tumors of the lungs* we may mention *carcinomata*, *melanomata*, *sarcomata*, *cavernous tumors*, *chondro-adenomata*, and *fibromata*. These neoplasms escape, as a rule, a clinical diagnosis; it is extremely difficult to differentiate these from simple indurations of the lungs.

Metastatic tumors may probably be diagnosed. The pulmonary symptoms which they produce, and the existence of external primary tumors (carcinomata), permit of the conclusion of a transfer of the neoplastic cells toward the organs of the pectoral cavity (circumscribed dull zones, dyspnoea, pulmonary phthisis, etc.). In a dog which was affected by primary carcinoma of the lungs we have seen frequent hemoptyses.

6. Metastatic or Embolic Pneumonia.

Etiology. This form of pneumonia represents also a secondary disease. It is developed when disintegrated solid products proceeding from a peripheric thrombus, or an open vein in the gangrenous centre, or from the heart (emboli), are carried by the blood and stop in the pulmonary bloodvessels. Enclosed in these latter the embolic masses produce at first hemorrhagic infarcts, and when they emanate from a purulent or septic centre provoke suppuration or pulmonary gangrene. In domestic animals its most frequent causes are phlebitis of the jugular, thrombophlebitis of the posterior legs (in the horse), gangrenous inflammations of the subungulated tissues, plantar cushion, etc. (Siedamgrotzky), purulent mastitis (Haase), abscesses of strangles, pyogenic processes developing in the different organs, ulcerous endocarditis, etc. In a horse Semmer has observed a fatty embolus of the lungs consecutive to an extensive purulent infiltration of the thigh.

Pathological anatomy. The alterations of metastatic pneu-

monia are disposed in centres (*lobular pneumonia*), the dimensions of which vary, as a rule, between those of a pea and a walnut. On account of the mode of ramification of the pulmonary bloodvessels they are found specially localized toward the surface of the lobes. Hemorrhagic infarcts of conical form, and the bases of which are directed toward the pleura, appear in the shape of hepatized and very dense islands of a dark-red color; when they contain pyogenic or septic elements they excite suppuration in their neighborhood; the infarct is gradually transformed into a purulent gangrenous island of greater or less extent and of a rounded shape (cavern). We recognize also the alterations of pyemia and septicemia.

Symptoms. Metastatic pneumonia is marked by an intense fever of sudden onset, by chills, dyspnoea, cough; in some cases we observe dulness and the ordinary symptoms of pulmonary cavities. It is almost always possible to discover the morbid centre which is the source of the infection. Generally death occurs by purulent infection. Recovery is extremely rare; it may, however, take place when the gangrenous centres are few and slightly extended. Eruption of the pus into the pleural cavity determines a purulent pleurisy (empyema).

The *treatment* is that of pulmonary gangrene. Its efficacy is very uncertain.

PULMONARY EMPHYSEMA.

Etiology and pathology. Emphysema is a disease characterized by extensive dilatation of the lungs and augmentation of the volume of air contained in that organ. Its mode of production is complex. In most cases it is determined by causes of a purely mechanical nature, the principal of which are: the abnormal enlargement of the thorax in the forced inspirations, an excessive dilatation of the lobulo-alveolar walls in certain parts of the lungs when air no longer enters into the atelectatic or inflamed portions, and violent coughing spells. It may also be produced by alterations of the lungs consecutive to phlegmasia of that organ (pneumonia, bronchial catarrhs) or connected with advanced age (senile emphysema). In some subjects there seems to exist a predisposition of the pulmonary tissue to the degenerative process (rarefaction of the interalveolar walls). Certain gaseous products of decomposition (hydrogen, carburetted hydrogen, hydrosulphuric acid) set free during the course of pneumonia by foreign bodies and contagious

plenro-pneumonia may also occasion it (septic emphysema). Emphysema consecutive to pulmonary traumatism belongs to the domain of surgery.

We must distinguish in emphysema the following three forms:

1. CHRONIC OR SUBSTANTIAL EMPHYSEMA (vesicular emphysema). This variety is characterized by dilatation of the alveoli and lobules, and by atrophy of interalveolar walls. These modifications are produced by a permanent augmentation of the air-pressure, which is followed by degenerative alterations of the pulmonary parenchyma. The much dilated alveoli become confluent and constitute cavities of greater or less extent. The more they are dilated the thinner their walls become. Measurements made by Stömmer upon the lungs of emphysematous horses have shown that the dimensions of the alveoli are ten times greater than in the normal lungs, where the diameter is about 0.15 mm. The diminution of the thickness of the alveolar walls is in proportion to the amplification (of 8μ in the healthy lung, the thickness of the walls decreases to $\frac{1}{2}\mu$). Rarefied walls are presented with very thin lamella which protrude more or less. The vascular system of the lungs undergoes remarkable changes. Alveolar ectasia determines a widening of the meshes of the capillary plexus, the bloodvessels of which must necessarily become elongated; on the other hand, their permanent compression (increased intralobular pressure) leads to contraction and obliteration of these same canals. As soon as emphysema is developed the pulmonary capillaries form rectilinear figures, their calibre is diminished, and many are atrophied or destroyed. Next to the rounded vascular stumps which are produced by a rupture of the capillaries, we see others, which are slim and joined by very thin cords (vascular atrophy in the thickness of the walls). This vascular alteration immediately precedes atrophy of the parenchyma: the elastic fibres disappear, the epithelial cells undergo fatty degeneration. As macroscopic alterations we find the emphysematous pulmonary portions swollen, pale, soft, and elastic. The pulmonary lobes are rounded upon their edges, their surface shows depressions which are produced by the ribs, and they retain the imprint of the finger. On opening the thorax the lungs do not collapse, as in the normal state; neither do they become depressed when incisions are made into them. There exists besides a compensatory cardiac hypertrophy, which is a consequence of atrophy of the pulmonary capillaries.

The *relations of emphysema of the horse with broken-wind* have been cleared up by Stömmer. In the first edition of this work we insisted upon the necessity of an anatomical examination of the lungs in all cases of broken-wind. Dieckerhoff, relying upon theoretical considerations, has claimed that pulmonary emphysema did not play any rôle in the pathology of broken-wind, and that this was produced by a paralysis of the bronchial muscle (Reisessen's muscle).¹ Stömmer has offered facts to disprove this theory. In nine horses which were affected with pulmonary broken-wind this author constantly found chronic emphysema, a fact confirming the opinion which has already been expressed by Gerlach, Haubner, Bruckmüller, etc., that pulmonary emphysema is the principal cause of broken-wind; in the horse it represents, so to speak, a *disease which is produced by work* (in draught and running horses). Stömmer has also shown that the lungs of the horse are much more adapted to the study of emphysema than those of man.

2. INTER-VEVICULAR OR INTERSTITIAL EMPHYSEMA is a form which is quite frequent in domestic animals. It is determined by abnormal intralobular pressures and is characterized by a rupture of the walls, which is followed by an irruption of air into the interlobular connective tissue and in the lymphatic spaces. In most cases this emphysema is observed upon the surface of the lungs, immediately below the pleura (subpleural emphysema), which is sometimes detached from the parenchyma and forms pseudo-vesicles, the dimensions of which vary between those of a pea and a nut (bullous emphysema). These projections may be easily displaced by compression, contrary to what takes place in vesicular emphysema. From the subpleural spaces the air generally progresses toward the root of the lungs and then penetrates between the folds of the mediastinum; it may reach the entrance of the thorax, and spreads in the subcutaneous connective tissue of the shoulders and of the various superficial regions of the body, including the ears and tail (Martin).

This inter-vesicular emphysema, which is followed by subcutaneous emphysema, is frequent in the ox and the horse. Almost the entire bibliography which is given at the end of this chapter [see original work] relates to this form of the disease, which is scarcely mentioned in special works. Its usual causes are excessive exertions, violent tractions (Köhne) during very hot or very cold

¹ Dieckerhoff: Adam's Wochenschr., 1884.

weather, or when the animals go against the wind, exhaustive running [runaway horses (Haussmann)], shipping horses long distances (Maris), violent coughing spells during work, and cough which is produced by irritating drinks (in the ox), administration of therapeutic liquids which contain acute or cutting vegetable particles which miscarry (Krüger, Eletti, Rivolta, Martini, Giles, Göring, Zalm). Schmidt has related a case of emphysema suddenly produced in a cow, which having fallen into a ditch was forced to make violent exertions in order to rise. Anacker has observed a similar case in an ox which had resisted the adjustment of the appliances necessary to perform an operation, and Demarchi has seen a heifer affected by emphysema after having bellowed for two days almost continuously.

Circumscribed vesicular emphysema is sometimes a lesion of suffering, which is also produced here by an augmentation of intra-alveolar pressure.

Under the name of *Pneumatoxis of the ox*, Michels has described a pulmonary emphysema which exists in an enzootic state in the swampy regions of the Netherlands, the pathology of which is absolutely unknown. Demeester, who has observed the disease in an enzootic state in Western Flanders (Belgium), attributes it, not without reason, to a bronchial catarrh which is accompanied by violent coughing, and due to taking cold while at pasture. Other Belgian veterinarians have also mentioned it. Littinger has recognized it in fattening stables in the neighborhood of Metz. At the autopsy he found vesicular, interlobular, and subpleural emphysema. This author attributes the disease to cold, which acts upon animals accustomed to remaining in stables of a high temperature.

3. ACUTE VESICULAR EMPHYSEMA. This is a simple alveolar ectasia without any anatomical alterations of the walls. It is sometimes diffuse, in other instances it may be limited to certain lobules. In some cases acute diffuse vesicular emphysema is a lesion of suffering, in others it represents the initial stage of interlobular emphysema. Partial vesicular emphysema occurs frequently as a collateral lesion when certain parts of the lungs have become impenetrable to air (chronic bronchial catarrh). The affected lobules are distended, more or less prominent upon the surface of the organ, and very pale. This form of the disease is of no clinical importance.

Symptoms. 1. The symptoms of *chronic or substantial em-*

physema are those of broken-wind. We observe a permanent dyspnoea, which is especially marked at the time of expiration, the factors of which are, on the one hand, a diminution of pulmonary elasticity and stagnation of air in the dilated alveoli (expiratory dyspnoea); on the other hand, atrophy of the capillary system of the lungs [want of oxygen in the blood on account of a contraction of the breathing surface (inspiratory dyspnoea)]. During the course of emphysema the heart generally undergoes a compensatory hypertrophy. (Concerning the other symptoms of this form of emphysema, see Broken-wind.)

2. In acute *interlobular emphysema* the animals are suddenly seized with intense dyspnoea and attacks of suffocation; the physiognomy is anxious, the respiration painful and plaintive. In the ox the mouth is half open and the tongue hanging. The abdominal muscles are very active (expiratory dyspnoea). There is at times a short, weak, and painful cough. Percussion gives a strong sound, with tympanitic resonance. On auscultation we hear crepitant, quivering blowing bruits and dry râles. Appetite and thirst are absent, the peristaltic movements are wanting; the temperature remains normal.

This emphysema has sometimes a subacute course. Death may be produced by asphyxia within twenty-four to thirty-six hours. In other cases where the process has a slower evolution, recovery occurs within a time varying from several weeks to a couple of months.

The *treatment* is limited to cases where there is subcutaneous emphysema; then we must give exit to the air by incisions made upon the skin. We may also resort to massage of the emphysematous regions and to their compression by means of bandages.

BROKEN-WIND OF THE HORSE.

Definition. *Broken-wind* is a symptomatic apyretic morbid condition, which is ordinarily incurable; its dominating symptom is "chronic dyspnoea." It rarely leads directly to death, but horses affected by it soon become incapable of performing the exertions required in their different employments. It is most frequently observed in adult or aged horses (over six years old); nevertheless, the ox, dog, and other domestic animals are not exempt from it. This trouble has also been given the names of *tight chest*, *abdominal*

and *cardiac respiration*, *coarse respiration*, and *cardiac broken-wind*. The expression broken-wind has rather a *legal* than a *clinical* meaning. However, here we shall consider it especially from the latter standpoint. (Concerning broken-wind, a disease considered as setting aside a contract of sale, see Gerlach's *Treatise on Legal Veterinary Medicine*.)

Etiology. Broken-wind may be determined by very varied pathological conditions. The principal are :

1. *a.* Diseases of the lungs and particularly chronic vesicular emphysema. *b.* Chronic bronchial catarrh and the alterations which complicate it : bronchiectasis, atelectasis, pulmonary sclerosis, partial emphysema, peribronchitis. *c.* Capillary bronchitis (bronchiolitis) and its complications. In this disease dyspnœa is produced by a contraction of the bronchi, by a lessening of the extent of the respiratory surface (inspiratory dyspnœa), and by a weakening of the elasticity of the lungs, a consequence of the chronic alterations of this organ (expiratory dyspnœa); this latter factor plays a leading rôle in the majority of cases. *d.* Tumors of the lungs : sarcoma, carcinoma, glandered neoformations, etc., adhesion of its lobes to the pectoral walls, connective induration and atrophy of the pulmonary tissue after pneumonia.

2. *Contraction of the respiratory ducts* (nose, back of the mouth, larynx, trachea), which is produced by thickening of the mucous membrane, by polypous neoformations, by bony alterations, by compression exerted upon the laryngo-tracheal duct (goitre), by cicatricial processes (tracheotomy), by paralysis of the dilators of the glottis—a lesion which is accompanied by chronic wheezing.

3. *Diseases of the heart*, especially valvular lesions, dilatations and anomalies of the inter-abdominal wall (Csokor); these diseases determine circulatory troubles in the lungs and cardiac *broken-wind*.

4. Compression of the lungs in cases of hydrothorax, diaphragmatic hernia, hypertrophy of the liver or spleen. We consider these influences as causes of broken-wind, for the respiratory troubles which they produce are ordinarily chronic, apyretic, and incurable.

The existence of *nervous* or *remittent broken-wind* is at least doubtful. It is generally admitted that it consists of a "chronic spasmodic dyspnœa," which is interrupted at regular intervals. This form of broken-wind would thus be identical with *chronic*

asthma of man. The numerous hypotheses which have been offered as to the causes of this latter testify that the term asthma is, like the word broken-wind, a generic expression. Asthma has been considered as being due: 1. To a spasm of the bronchial muscle. 2. To diaphragmatic spasms. 3. To acute exudative bronchiolitis, which is manifested by attacks. 4. To a vaso-dilator neurosis, which is accompanied by intense hyperemia of the bronchioles, thus causing a contraction of their opening. In this form of asthma, which is probably the most important, the attack is produced in a reflex manner: the respiratory nerves possess a hyper-excitability which is found in certain pathological processes of the nasal and guttural cavities—polypi, mucous uceformations, hyperplasia of the turbinated bones—and which would play the rôle of predisposing influence. In some subjects reflex asthma is also produced by the inhalation of hay dust (hay asthma, which is said to have been observed in the horse), by certain olfactory sensations (odor of rose, violet, etc.), by extreme temperatures, after the absorption of certain medicinal agents (salicylic acid, sulphur), and after cerebral excitement. It is observed especially during particular periods and in certain stables. There have been described a dyspeptic, uremic, diabetic, syphilitic, and saturnine asthma.

In all these varieties the existence of an abnormal individual irritability, which interferes as a predisposing cause, is admitted. It is possible that there exists in the horse and other domestic animals similar respiratory spasms—that is to say, different forms of asthma; but this question can only be solved by new researches.

Symptoms. The essential symptom of broken-wind is dyspnœa. It may be manifested in various ways:

1. Respiration is accelerated; after five minutes' exercise at a rapid gait we may count from fifty to sixty respirations and more per minute (instead of about thirty); if the gait is rapid and the exercise prolonged for half an hour to an hour, there are from eighty to one hundred respirations (instead of fifty). The movements of the ribs are usually very limited.

2. Respiration is more or less laborious. Expiratory dyspnœa is especially pronounced. It is produced by a weakening of the pulmonary elasticity, and is apparent by an extraordinary activity of the abdominal muscles, which, as a rule, become contracted by a double movement; the first is short, the second is longer; these peculiarities are distinctly marked on the surface of the abdomen,

where the muscles outline the furrows of broken-wind. The flank is the seat of movements of lowering and rebounding, which are very pronounced and very abrupt (*subsultus*); the anus, which is thrown back, forms a projection of greater or less size. The duration of expiration is much greater than that of inspiration. Inspiratory dyspnoea is characterized by an abnormal rising of the ribs, by an extreme dilatation of the nostrils, and, in case of chronic wheezing, by bruits of contraction. It is caused by a diminution of the breathing surface and various alterations which hinder the entrance of air into the respiratory ducts.

3. Respiration becomes slackened to extreme slowness. In a normal state it returns to its physiological rhythm in ten to twenty minutes after half an hour's exercise. In a broken-winded horse this does not take place until after a half hour, an hour, or sometimes a still longer lapse of time.

In a state of rest broken-winded horses are affected with a short, weak, abortive cough, which is accompanied by a discharge. Percussion and auscultation do not ordinarily reveal any abnormal phenomenon. However, in quite a large number of broken-winded subjects we have recognized a manifest lessening of cardiac dulness (the heart is covered by the emphysematous lungs). In the so-called cardiac broken-wind (related to a cardiopathy) we observe with dyspnoea symptoms of heart troubles: the precordial impulse is usually very strong—sometimes it is imperceptible; the pulse is accelerated (80 to 120 pulsations per minute), irregular, and generally weak. When a valvular affection exists we perceive modifications of the heart sounds or abnormal bruits.

The animals perspire on the slightest exertion, and sometimes abundantly; nutrition is poor; the hair is dull and bristly.

Lustig considers as a diagnostic symptom of broken-wind the appearance of albumin in the urine after exercise. We do not attach any importance to this albuminuria; we have observed it after work in horses which were in perfect health, and it may be wanting in subjects which are affected by very marked broken-wind. When albuminuria exists it is explained by a diminution of the filtrating pressure in the renal glomerules. Siedamgrotzky has also called attention to the increase of the proportion of oxalate of lime and other mineral substances in the urine after exercise, an increase specially noticeable in broken-winded horses; this peculiarity may also be explained by a lessening of the filtrating pres-

sure: the proportion of salts and albumin which pass through the renal filters is diminished, that of water is augmented. Phenomena of this kind may also be produced in a large number of other diseases.

Broken-wind has a chronic *course*. It presents quite frequently partial remissions and aggravations corresponding with the season, weather, alimentation, and work. It is extremely variable in its intensity; in certain benign cases it affects the sick animal's usefulness but slightly; in others, when very serious, it renders it unable to perform any service, and in practice we may meet with all possible gradations between these extreme forms.

Treatment. The medicinal agent most used is arsenious acid administered in small doses for a long time. At present it is impossible to give an interpretation of the mode of action of this drug; no precise research has been made in that direction. Assertions of authors concerning its efficiency differ widely. Some claim that it produces a great improvement, others consider it as of absolutely no effect; others, in fine, affirm that it is hurtful. Results of experiments made upon animals have established the fact that arsenious acid administered for a long time increases nutrition and flesh; but its influence in emphysema and dyspnœa remains to be demonstrated.

We will omit the consideration of the entire list of remedies used in this disease. It is advisable to give as small a quantity of water as possible; this has already been recommended by Garsault.¹ We know that suppression of drinks plays an important rôle in the modern treatment of chronic circulatory troubles (Oertel). The heart acts so much easier in proportion to the smaller quantity of organic liquids. This indication must be observed especially in cardiac broken-wind, and in all forms of the affection which accompany circulatory troubles.

When broken-wind coexists with chronic bronchial catarrh we may use alkalines in the form of artificial Carlsbad salts (sulphate of soda, 80 grammes; chloride of sodium, 15 grammes; bicarbonate of soda, 5 grammes—to be administered three times daily). Whatever may be the degree of intensity of broken-wind, it is advantageous to give easily-digested and nutritious food and practise hygienic care in all cases.

Broken-wind is considered a disease setting aside a contract of

¹ Garsault: Parfait mareschal. Paris, 1755.

sale in Bavaria, Württemberg, and the Grand Duchy of Baden (delay fourteen days), in Saxony and Austria (fifteen days), in Switzerland (twenty days), in the Duchy of Brunswick and in Prussia (twenty-eight days).

VI. DISEASES OF THE PLEURA.

Etiology. Inflammation of the pleura is *primary, idiopathic*, or *secondary*, and is related to some pre-existing disease.

It is *simple* or *double*, according to its localization on one or both pleural pouches. Unilateral pleurisy is more frequent than double pleurisy.

Primary pleurisy is especially observed in the horse and the dog. In a great many cases it is produced by *cold* (rheumatismal pleurisy). The origin *à frigore* of this disease is established by certain observations. In a flock Duviensart observed pleurisy in 200 sheep which had been shorn during the winter (in the month of February). But it is very far from being true that rheumatismal pleurisy is the consequences of cold in all cases. Frequently it appears to be the result of other atmospheric influences or of specific causes.¹ Very young animals seem to be predisposed to it. Immelmann has seen 200 sucking lambs out of a total of 600 die from pleurisy. Nevertheless it does not spare vigorous adult animals (draft horses); in these it seems to be produced by laborious exertions while exposed to cold.

Secondary pleurisy occurs as a complication of a great variety of diseases. It is commonly developed in the course of specific pneumonia (contagious pneumonia of the horse, contagious pleuro-pneumonia). Croupous and catarrhal pneumonias of the ox, pulmonary gangrene, and neoplasm of the lungs are also very frequently accompanied by it, and tumors of the mediastinum oftentimes maintain the disease in a chronic state. We have observed a case of acute

¹ Some cases have been reported which tend to establish the existence of infectious pleurisy in domestic animals. In a regiment of cavalry Humbert has seen three horses die from acute pleurisy which had entered the infirmary successively for accidents which were of a traumatic nature, and that had been placed in the same stall. The disease was characterized by the classical symptoms and alterations; in one case they observed soon after the invasion an abundant epistaxis, which persisted for forty-eight hours. In these three cases the place which the patients occupied in the infirmary enabled them to exclude the action of cold as a cause of the disease (see *Receuil Vét.*, 1887).—N. D. T.

pleurisy determined by an extensive glanderous abscess which was developed in the mediastinum. Jabots, tumors, and wounds of the œsophagus often occasion pleurisy of a gangrenous character. In the horse and the dog we have found several cases of gangrenous pleurisy which were produced by a needle, bone, or other foreign body that had perforated the œsophagus. Inflammation of the pericardium, especially when determined by a foreign body, may be propagated to the pleura in the same way as that of the peritoneum may also pass to the thoracic serous membrane through the diaphragm. In some cases pleurisy is but an incident of inflammatory or suppurative diseases of the bones of neighboring regions—sternum, ribs, vertebra. *Traumatic* pleurisy is another secondary form, which is consecutive to contusions of the thorax, to penetrating wounds of the chest, to fractures of the ribs, to thoracentesis, to the action of foreign bodies (spears of grain) falling into the bronchi. We see also the appearance of metastatic pleurisy in pyemia, septicemia, acute articular rheumatism, and chronic nephritis. Finally, pleural inflammation is a frequent epiphenomenon of tuberculosis and glanders.

Pathological anatomy. From an anatomical point of view we recognize in pleurisy the *circumscribed, diffuse, dry, exudative, and adhesive* forms. According to the characters of the exudate, it is *fibrinous, sero-fibrinous, serous, purulent, hemorrhagic, or gangrenous*.

In the beginning of the phlegmasia we find the subpleural blood-vessels and lymphatic canals of the pleura much injected. The serous membrane is dull, opaque, and often studded with hemorrhages; soon it becomes covered with very fine yellowish-white fibrinous flakes, which are slightly adherent, and are composed of fibrine, detached epithelial cells, white corpuscles, and cellular nuclei. The persistence of the process to this degree of intensity and the slight exudation, which is the result of it, characterize *dry pleurisy*. When the exudate does not become absorbed the white corpuscles are transformed into fibroblasts, afterward into connective-tissue cells, and the new-formed layer soon constitutes a very vascular granular tissue, which becomes indurated and contracted; the pleural folds, which are thickened, are partially adherent (adhesive pleurisy). In cases where the inflammation is circumscribed the serous membrane shows small whitish spots (milky spots), also neoformations having the appearance of pimples and fibrinous filaments which bind the lungs to the costal wall; when it is diffuse

we remark extensive membranous growths and thickening of the pleura. In very old cases of pleurisy the serous membrane is covered with dense layers several centimetres in thickness, and which are formed by callous, sometimes calcified, connective tissue. These lesions are always the result of the existence of certain acute phenomena.

In cases of *exudative pleurisy* the sero-fibrinous form is the most frequent. A relatively abundant quantity of liquid may be poured out in the space of a few hours. In the horse there is, as a rule, from 20 to 50 litres of liquid in the two pleural pouches; Holmes has found as high as 75 litres. In the dog the average quantity is from $\frac{1}{2}$ litre to 4 litres; in the pig from 2 to 10 litres. According to the case, the exudate is of a yellowish, reddish-yellow, or greenish-yellow color; it holds in suspension fibrinous, flaky, or lamellar clots; when it is in process of absorption the serum disappears first; the fibrinous productions can only be renewed after having been modified by fatty or mucous degeneration; sometimes they become encysted or undergo caseous transformation. A purely serous exudative form is very rare.

Purulent pleurisy (empyema) occurs after penetrating wounds of the thorax in the course of certain infectious diseases or processes which are accompanied by metastasis (pyemia). The exudate is principally formed of white corpuscles; its absorption is exceptional. In some cases the pus escapes externally by way of the bronchi and afterward the air penetrates into the pleural cavity (pyopneumothorax); in other circumstances the purulent matter perforates the pectoral wall and escapes (*Empyema necessitatis*). When the pus is thus enabled to escape the empyema may be cured; the purulent cavity becomes filled with granulations by the formation of fibrinous tissue.

Pleurisy assumes a *gangrenous* type when the air has access to the pleural pouch through either the bronchi or the thoracic walls, but especially when abscesses or pulmonary cavities are opened in the pleura. This form of the disease is quite frequently found during the course of gangrenous pneumonia and also contagious pneumonia. It is characterized by a fetid exudate of bad aspect, which holds in suspension numerous products of decomposition.

The *hemorrhagic form* is observed in cases where pleurisy is accompanied by the "hemorrhagic diathesis," in those where the absorption of the fibrinous exudate is not very active, or when an

abundant vascular neoformation is produced in the fibrinous masses. The exudate, which is of a reddish-brown color, is filled with red corpuscles.

As soon as the pleuritic liquid exists in abundance the lungs are compressed in the superior portion of the pleural cavity, lessened in volume, increased in consistency, and become atelectatic. The healthy pulmonary regions are hyperemic and emphysematous.

The heart is deviated from its normal position; the diaphragm is thrown back toward the abdominal cavity; the large thoracic bloodvessels are compressed—a phenomenon which explains the blood stagnations produced in the venous system (passive congestion of the liver, kidney, etc.).

Deformity of the thorax, consecutive to pulmonary atrophy, is a slow lesion of unilateral pleurisy.

Symptoms. Rheumatismal pleurisy ordinarily starts with chills and a marked increase of the general temperature; in some cases there are other rheumatismal symptoms: colics, pain during movements, etc. Fever, which is quite high in the beginning (41°), falls considerably later on (40.5° – 39.5°); it is distinguished from that of pneumonia by its atypical and intermittent course; quite frequently we may observe stages of apyrexia which are prolonged for several days. As a rule, the circulation is much accelerated—this condition is probably due to the pressure exerted by the exudate upon the heart. In the horse we count from 60 to 100 pulsations per minute. The pulse is almost always small, scarcely perceptible. The patients are very weak, the appetite is absent, the head is often rested upon the manger; it is not rare to find the temperature on the diseased side higher than on the healthy side; the conjunctivæ are injected. In some cases the disease has a slow, chronic evolution, and we may be surprised to detect a much extended dulness during the first examination.

Respiration has undergone characteristic modifications. It is accelerated, laborious, and especially abdominal. The movements of the thoracic walls, which are very limited, contrast with those of the flank, which are much more extended (beatings of the flank). When the exudate is very abundant respiration is heavy and *discordant*; at the moment of inspiration, when the ribs are rising, the flank is hollow; it becomes filled or inflated when the ribs are lowered; the entire trunk is swung from one side to the other; in

some cases the front legs are spread, in other instances one is placed in front of the other. The nostrils are much dilated; discharge is wanting; generally there is a laborious, short, painful cough. The patients utter groans when they are forced to change their position, especially where the intercostal spaces are compressed; they lie down very rarely, and then almost always upon the healthy side.

A physical examination of the chest is of the highest importance from a diagnostic standpoint.

On inspecting the thorax we recognize in some cases an enlargement of the chest on the diseased side; the intercostal depressions are effaced or replaced by slight projections. The precordial impulse is sometimes displaced; in pleurisy located on the left side palpation finds a deviation of the heart; this organ no longer beats on the left side, but—especially in young animals—its action may be easily perceived on the right side.

As long as the level of the pleuritic liquid does not rise above the lower third of the chest, percussion could not furnish any information in the horse. When the exudate is more abundant it produces dulness, which is limited above by a horizontal line; this is a characteristic symptom when the dull zone varies with the position given the animal (dorsal decubitus, elevation of front and hind quarters), but a displacement of the liquid is often slow or incomplete, and in some cases does not take place. On percussion of the exudate we notice also a more marked resistance of the pectoral walls under the pleximetric hammer.

We recognize sometimes a tympanitic sound during the first period of the disease, when the small quantity of the exuded liquid has diminished the elasticity of the lungs. This same tympanitic sound also exists above the line of dulness, and this is accounted for by the fact that the lungs, which have lost their elasticity, are pushed back and compressed by the exudate. The dulness never exceeds in area more than two-thirds of the chest.

In the beginning auscultation frequently enables us to recognize pathognomonic friction bruits (filing, quivering, and gliding sounds, which are produced in a jerky manner), which are soon replaced by liquid bruits, to reappear again toward the close of the disease. These bruits, which are sometimes perceptible to the hand, are not constant; they are wanting, even during the initial stage, in all cases of pleurisy which are of a distinctly exudative character. The vesicular murmur becomes more and more weak in proportion

to the increase of the exudate, and it ends by disappearing completely. Bronchial wheezing (tubal wheezing) replaces it when the lungs are slightly compressed; but when the very abundant exudate makes its action felt upon the bronchi the wheezing disappears in its turn; in some cases it exists above the line of dulness during expiration as well as inspiration.

When the pleurisy is unilateral on auscultating the healthy lungs we hear a more intense vesicular murmur, except in serious cases, where abnormal bruits produced by pulmonary œdema are perceived (crepitation râles). We may also recognize a dull, muscular bruit, which is caused by the contractions of the intercostal muscles.

Changes which take place in the urine deserve to be mentioned. In proportion to the augmentation of the exudate, the quantity of urine passed diminishes, and a time arrives when it no longer contains chlorides; later, when the exudate is absorbed, it again becomes secreted abundantly and chlorides reappear (critical urinary secretion). Besides, the urine is frequently albuminous. In the horse, according to our personal observations, it often preserves its alkaline reaction for a long time, contrary to indications which are given by most authors.

The symptoms we have just detailed are only observed in extended pleurisy. Dry circumscribed pleurisies are often not marked by any important morbid phenomena.

Course and termination. In a general way the prognosis of pleurisy is more serious than that of croupous pneumonia; but in recent rheumatismal pleurisy it is generally favorable. In a large number of cases absorption takes place very rapidly and completely. Extended pleurisies may, however, determine rapid death by pulmonary œdema, by arresting the action of the heart, or by asphyxia. The disease often pursues a dragging course which is interrupted by a return of the trouble. Convalescence is slow. False membranes with which the lungs are frequently covered and adhesions between the pleural folds determine persistent respiratory troubles. There are cases where pleurisy assumes a chronic type with numerous spells of acute wheezing. The exudate displaces the organs; it compresses them and determines circulatory troubles (heart and venous system), also dropsies, progressive emaciation and cachexia. Lastly, there is a possibility that the exudate may become purulent. As soon as empyema is established it is marked by repeated chills,

an intense hyperthermia, symptoms of hectic fever, profound collapse, and is often complicated by pneumothorax (pyopneumothorax).

Diagnosis. The principal diagnostic symptoms of pleurisy are: friction bruit, regular and complete dulness, increased resistance of the pectoral walls under the action of the pleximetric hammer, sensitiveness of these walls to percussion, abdominal type of respiration, disappearance of the heart impulse and its bruits, absence of nasal discharge, and the atypic and irregular course of the fever. In the beginning it is frequently difficult to differentiate pleurisy from pneumonia. In such cases it is advisable to repeat the examinations on the same day in order to detect the friction bruit, which has ordinarily a very ephemeral duration. (See Pneumonia.) In doubtful cases we may resort to an exploratory puncture.

Treatment. In all its phases pleurisy requires active treatment. It may be combated by various means. In the beginning refrigerant compresses upon the thorax and cold irrigations are of advantage. The horse may be placed directly under a hydrotherapeutic apparatus. Cutaneous revulsives applied upon the diseased side have an action similar to that of cold water. In the horse we use either sinapisms or frictions of diluted essence of mustard (essence of mustard, 1; alcohol 12–20, or essence of turpentine, ether, etc.); under the influence of these agents hyperemia is lessened, the temperature lowered, and the pains diminished.¹ These frictions have the inconvenience of rendering percussion impossible for some days. For the fever we should employ the antipyretics generally used: thalline, antifebrine, phenacetine, antipyrine, etc., but we especially use digitalis, which acts at the same time as a diuretic.²

¹ Brunet advises resort to a *graded revulsion* which is obtained by applications of a vesicant mixture having as a formula:

Basilicum	500 grammes.
Pulverized cantharides	100 “
Euphorbia	50 “
Croton oil	10 “

These applications are made in an upward direction over the whole extent of the thorax, and upon a width of ten to fifteen centimetres.—N. D. T.

² Salicylate of soda, which was advised by Aufrecht as early as 1883 in order to combat serous pleurisy in man, has produced good results in the hands of Talamon and several foreign physicians. It is given in a dose of 4 to 6 grammes daily. Besides its diuretic action, this agent appears to exercise a direct influence on the inflamed pleura. Experiments of Rosenbach and Pohl have demonstrated a penetration into the serous cavities of the salicylate which is introduced by the digestive tube. This medicament deserves a trial in domestic animals.—N. D. T.

When absorption of the exudate takes place slowly we may accelerate it by a dry diet, diuretics, drastics, sialagogues, and diaphoretics.

Among diuretics we use, as a rule, digitalis (for the horse 5 to 10 grammes per dose; for the dog 0.1 to 0.2 gramme, also alkalies, and especially acetate of potash, juniper berries, or essence of turpentine. Recent observations have established that calomel, which was formerly used as a purgative, also possesses diuretic properties. Trasbot advises the administration of this agent in a dose of 2 grammes per day; he advises also applications of vesicating ointment upon the thorax. As a sialagogue, pilocarpine in subcutaneous injections (horse, 0.1 to 0.2 gramme of hydrochlorate of pilocarpine; for the ox, 0.2 to 0.4 gramme; for the dog, 0.005 to 0.02 gramme) has been recommended. A simple compress of Priessnitz may hasten absorption.

In certain cases we should practise thoracentesis. This operation, which is of great importance, is absolutely devoid of risk when it is performed according to the rules of modern surgery. We must resort to it: 1. When the quantity of exudate is such that asphyxia is imminent; it is here *indicatio vitalis*. 2. In cases where the exudation is abundant. 3. Where the exudate persists without any tendency to absorption. 4. In purulent and gangrenous pleurises. These latter cannot be healed by puncturing only; they necessitate incision of the thorax and drainage of the pleural cavity. The wounds produced on the lungs are not alarming; they have rarely any serious consequences. Iodated injections, which were formerly used quite frequently, have been abandoned. (For information concerning this operation, see the *Traité de Médecine Opératoire*.¹)

¹ Thoracentesis may be performed either with an ordinary trocar or with a hollow capillary needle and Dieulafoy's exhauster. The latter method gives good results. It is best to use a No. 2 needle of a calibre of 1.2 millimetre. The apparatus should first be exhausted, the needle is then adjusted on the rubber tube and introduced into the place selected to a depth of 2-3 centimetres; then opening the corresponding stop-cock of the exhauster, the liquid flows into the receiver. If the needle becomes obstructed by a fibrinous clot, the obstruction can be removed by a stroke of the suction rod. As soon as the receiver is filled, close the stop-cock which puts it in communication with the pleura, and open the one through which the liquid is discharged. The operation is repeated till a sufficient quantity of liquid has been abstracted (an average of 15 to 20 litres). If the operation is performed in this way it is absolutely harmless. The small calibre of the needle assures the slow unfolding of the lungs, and we have to fear neither asphyxia nor other complications which were liable to happen in the old method. Thoracentesis, in conjunction with vesicant frictions

DROPSY OF THE CHEST: HYDROTHORAX.

Etiology. Hydrothorax is observed in all domestic animals, but especially in the dog. Though it constitutes a secondary disease, it is usual to devote a special description to it. It has also been designated *chronic dropsy of the chest*, in contradistinction to *inflammatory dropsy*, formerly synonymous with pleurisy. Hydrothorax may depend upon a general dropsy consecutive to various alterations of the blood, to a greater penetrability of the blood-vessels, to the conditions which accompany chronic diseases of the lungs, heart, kidneys, and particularly valvular diseases (we have observed such a case in the horse). It is not rare to witness the coexistence of hydrothorax, hydropericarditis, and ascites.

Pathological anatomy. Alterations of hydrothorax are marked in both pleural pouches. The latter contain a clear, yellowish or reddish serous liquid, which is free from fibrinous flakes. The pleura is smooth, infiltrated, and slightly softened by the exudate. The lungs are compressed, the diaphragm is thrown backward, and the intercostal muscles are crowded outward.

Symptoms. Hydrothorax does not determine any febrile reaction. Its principal symptom is *dyspnoea*. Respiration, which is more or less laborious, retains the costal type. Dulness is limited above by a horizontal line, the situation of which varies with the position given to the animal. The dull region sometimes gives no response on auscultation, but immediately above we may perceive bronchial wheezing (tubal blowing). We recognize, besides, symptoms of the primary affection; it is mostly chronic valvular endocarditis (abnormal bruits, indistinct or confused sounds). In some instances critical examination of the patients reveals the ex-

made upon the thorax, constitutes a treatment which has given good results in the horse. Chuchu, who has used it for ten years, succeeded in saving nineteen patients. Of late years Hoffmann and Ughi have tried intra-thoracic medicated injections and flushings of the pleura. In order to flush the serous membrane we may use solutions of chloride of sodium, of sublimate (at 1:1000), phenic acid, salicylic acid, thymol (Hoffmann), or sulphophenate of zinc (Ughi)—the solutions having a temperature of 38–39° C. After puncturing the thorax and extracting a certain quantity of liquid, a rubber tube is adjusted by placing one of its ends to the trocar and the other to a vase containing the medicated solution. This should be introduced into the pleura by placing it above the level of the thoracic cavity; it is extracted by lowering the vase or by means of an exhaustor.—N. D. T.

istence of other dropsies. A recognition of the exudate by thoracentesis confirms the diagnosis.

Differential diagnosis. Hyperthermia, cough, and most of the symptoms produced by pleural inflammation (friction bruit, sore condition of costal walls, etc.) are wanting in hydrothorax. On the other hand, pleurisy is frequently unilateral, and its exudate is turbid, fibrinous, and very rich in albumin; hydrothorax, on the contrary, is always bilateral; its exudate is free from fibrinous flakes, poor in albumin, and is much more easily and completely displaced than pleuritic exudate.

Treatment. This varies with the causal disease. We resort, as a rule, to a dry diet and to diuretics. In the dog we prescribe: infusion of leaves of digitalis, 1:140; solution of acetate of potash and fluid extract of juniper berries, each 25 grammes—1 to 2 teaspoonfuls or tablespoonfuls per day. In this animal pilocarpine has given us good results (0.005 to 0.02 gramme per dose, in a subcutaneous injection). Resort should be had to thoracentesis only when life is in danger. The exudate always rapidly returns to its normal condition.

PNEUMOTHORAX.

Etiology. Pneumothorax is a disease produced by the penetration of air or gas into the pleural cavity. When a pleuritic exudate exists at the same time there is *hydropneumothorax*, and if this exudate is purulent, *pyopneumothorax*.

Generally the air penetrates into the pleura through a pulmonary passage when favored by an abscess or lacerations of the parenchyma (interstitial emphysema of the ox). It reaches it more rarely through a perforation of the thoracic wall or a laceration of the œsophagus. In the ox the principal cause is perforation of the pleura by a vulnerating body which comes from the plexus. Certain foreign bodies in the tissues may penetrate into the lungs and cause pneumothorax (spears of rye in the dog—Weber).

Pathological anatomy. Pure pneumothorax is a very rare condition, and is only seen in interstitial pulmonary emphysema of the ox; the pleural cavity then contains atmospheric air; as a rule, its gaseous contents are poor in oxygen, but rich in carbonic acid and in carburets and sulphurets of hydrogen; sometimes we also find in it a serous or purulent liquid; the lungs are

often compressed, the heart deviated, and the diaphragm pressed backward.

Symptoms. Symptoms of pneumothorax ordinarily appear suddenly. We recognize an intense dyspnœa, a lowering of the general temperature, and symptoms of collapse. In cases where the disease is unilateral the corresponding half of the thorax appears dilated; percussion gives a tympanitic or amphoric resonance, and sometimes a sound similar to a broken-pot. When the air is much compressed the sound is clear and silvery. On auscultation we may observe a metallic tinkling, amphoric wheezing, and bruits of falling drops (which were observed in the horse by St. Cyr). In other cases we perceive a vague respiratory murmur or tubular blowing. Sometimes the thorax is absolutely silent.

Treatment. The air may be removed by means of puncturing, but in the majority of cases (pleuro-pulmonary perforation) the condition is soon reproduced. The symptomatic treatment consists in combating weakness by stimulants (camphor, hyoscyamine, caffeine, ether, and alcohol).

In man we distinguish three forms of pneumothorax: 1. *Open pneumothorax*, in which the air penetrates into the pleura through a large perforation. 2. *Ventilo-pneumothorax*, in which the air introduced into the pleural cavity partially closes the opening of the entrance; this gas enters freely, but only a small portion of it leaves the cavity. 3. *Closed pneumothorax*: in this form the lungs completely close the orifice through which the air has entered into the thorax.

ADDENDA TO DISEASES OF THE RESPIRATORY APPARATUS.

A. Non-parasitic Diseases of the Respiratory Apparatus of Birds.

In the different species of birds we observe a simple primary nasal catarrh, which is due to cold or to inhalations of irritating matter. It is generally designated under the name of "pip." The principal symptoms of this catarrh are sneezing, rattling or whistling respiration, a continual opening of the bill, slight dyspnœa, a continual shaking of the head, and a muco-purulent discharge, which exudes when slight pressure is made upon the nasal openings, it dries up frequently and forms thin crusts.

This disease is benign ; it is rarely necessary to institute treatment. In obstinate cases alkalies (Carlsbad salts) may be added to drinking-water. From a prognostic standpoint it is important to differentiate simple nasal catarrh from diphtheria.

Besides this rhinitis we may observe in the gallinaceous species forms of catarrhal and croupous tracheitis, bronchitis, and pneumonia.

Primary croupous pneumonia is of certain importance. According to Zürn, it is characterized anatomically by pulmonary hyperemia, hepatization, subpleural ecchymoses, serous extravasations, and by an accumulation in the bronchi of thick fibrinous productions. As symptoms we notice a difficulty of respiration, a permanent opening of the bill, cough, an orange-yellow discharge which comes out of the bill and the nasal cavities, and a soreness of the thoracic cavity.

B. Parasitic Diseases of the Respiratory Apparatus of Birds.

1. [EPI-ORNITIC W. L. Z.] DISEASE, WHICH IS DETERMINED BY THE SYNGAMUS TRACHEALIS (SYNGAMOSIS).

NATURAL HISTORY. This disease was observed in America in 1797, where it caused considerable damage. Its first description is due to Wiesenthal. In 1806 it appeared in England, where it has become, as also in France, one of the diseases which caused the greatest ravages in aviaries and breeding establishments for pheasants. In England it kills annually more than one million chickens. In France it has invaded the pheasantries of Rothschild, where it claimed more than twelve hundred victims daily (Mégnin).

The tracheal syngamus belongs to the family of strongylus. It has been observed in the pheasant, chicken, partridge, turkey, peacock, young goose (Prbylka), and many other birds : stork, woodpecker, starling, magpie, swallow, crow, jackdaw, etc. In a sexual state it is found in pairs, the male and female being joined in a permanent copulating way ; this parasite commonly locates in the upper region of the trachea ; in most cases it is found immediately below the glottis. It is of a red color and cylindrical form, and has a diameter of about 1 millimetre ; the female is 13 millimetres long and the male 5 millimetres ; both are fixed upon the bronchial mucous membrane by means of a suction cup ; they suck blood and

produce a tracheitis. Strong animals resist quite well; but in small species and young birds the trachea is sometimes entirely obstructed. Most of the birds die from asphyxia.

The eggs, which are elliptical and smooth, are thrown into the pharynx by the cough; from there they penetrate into the œsophagus and are ejected with excremental matter. Within eight days, when the temperature is high, and after several weeks when the weather is cold, these eggs produce anguilliform embryos, which are swallowed by healthy animals with the food; the course which they follow in order to reach the trachea is unknown. In infested animals the cough may appear after the seventh day.

Walker, having found the embryo of *syngamus* in the *lumbricoides* of those regions where the disease existed, has considered these worms as agents which transmit the disease. Salmon does not agree with this opinion; according to him, the *lumbricoides* contain the eggs of *syngamus*, because they swallow them by accident at the same time as the earth which contains them; but these worms would not be a necessary intermediate host for the parasite.

Be it as it may, Walker has the credit of having demonstrated that worms which are located in places where the *syngamose* exists may infest the birds by which they are eaten. This author recommends to destroy the *lumbricoides* in the soil of poultry-yards by means of salt water (?).

Symptoms. In the beginning the birds are less lively, they refuse food, and open the bill frequently as if to gape. Soon the cough appears, whitish mucus is ejected through the nostrils, and the head is shaken convulsively. When the frequency of the cough increases respiration becomes more and more laborious, the patients *snap the air*, they are affected by suffocating spells; in a large number of cases death occurs suddenly by obstruction of the glottis. The presence of eggs in the excrements is an important diagnostic symptom. Recovery may ensue in strong or old animals and in all cases where the *syngamus* are not numerous.

Treatment. The healthy birds must be separated from the sick (infection may be produced by direct ingestion of expectorated worms), the aviaries should be thoroughly disinfected, the cadavers burned, the food changed, and the troughs thoroughly cleansed: such are the principal prophylactic indications. Mégnin recommends adding garlic to the food and giving a decoction of this plant. We may succeed sometimes in extracting the *syngamus* by

means of a pair of pincers introduced into the laryngeal opening. In order to do this successfully Cobbold has practised tracheotomy.

Among other parasites of the respiratory apparatus of birds we must mention the *Monostomum flavum*, which is found in the trachea of the duck, and the *Filaria clava*, which is located on the interannular connective tissue of the trachea of the pigeon.

2. ACARIASIS OF THE AIR POUCHES OF THE CHICKEN (CYTODITES NUDUS).

NATURAL HISTORY. The *Cytodites nudus* or *Cytoleichus sarcopoides* is located in the trachea, bronchi, and air pouches (which, according to Leisering, are large alveoli), and especially the abdominal cavities.

To the naked eye the cystodites appear in the form of small white points, the diameter of which is about half a millimetre. Under the microscope they have the appearance of sarcoptiform acari; the body and the head are oval; they have four pair of legs with five divisions, and terminate in a pediculated suction cup.

Perhaps the *Cytodites nudus* and the *Symplectoptes cysticola*, which are found to be encysted in several organs of the abdominal cavity and in the subcutaneous connective tissue (see vol. i., page 548), represent two evolution stages of the same parasite. In several autopsies Holzendorff found these in the same regions.

The cytodites produce an intense croupous inflammation in the bronchi; the openings of these ducts are frequently obstructed by viscous masses of yellowish color; the bronchioles contain purulent mucus, which is mixed with bloody clots and very rich in parasites; the lungs are hepatized and œdematous in certain portions. We sometimes find the acari in very large numbers in the abdominal air pouches, which are at the same time free from inflammatory alterations.

Symptoms. The principal symptoms occasioned by cytodites are acceleration of breathing and dyspnoea. According to Zürn, the patients would utter particular cries which would lead to the belief in the presence of a foreign body introduced into the larynx. The general condition is not modified. Liveliness and appetite are preserved.

Treatment. This is of little efficacy. The principal indication is the use of acaricidal fumigations (tar or water of tar).

Edgar has found the *Cytodites nudus* at the autopsy of chickens which had died suddenly. The abdominal cavity was coated with a yellowish-gray dust which was composed of thousands of acari. He has also met with them in the pericardium, the heart, and in the aorta.

C. Pneumomycoses of Birds.

Mycoses of birds, which are found in the chicken, pigeon, goose, duck, swan, pheasant, parrot, and house birds, are produced by the *Aspergillus glaucus*, *A. nigrescens*, and *A. fumigatus*, as well as by the *Mucor racemosus*. They are not limited to the lungs; they involve also the bronchi, air pouches, and nasal cavities. Infection is produced by inhalation of dust or ingestion of mouldy food. Upon the mucous membranes the fungi produce lamellar or leaf-like diphtheric exudates. We find also purulent, hardened, caseous masses, which contain a large number of myceliums and conidies. In the lungs the alterations present a follicular type.

Their principal symptoms are: acceleration of respiration and a hoarse bruit which accompanies the expiration (Generali), diminution of appetite and extreme weakness (the wings hang and the eyelids are closed). Emaciation becomes rapidly accentuated, a profuse diarrhea appears, the patients become emaciated, and die within a few weeks.

SECTION IV.

CHRONIC CONSTITUTIONAL DISEASES.

WE give the name of constitutional diseases to morbid processes consisting either of anomalies of the blood or in troubles of the organic mutations or of nutrition. Some of these diseases (anemia, hydremia) belong to general pathology. We will specially study them from a practical point of view and by setting aside all theoretical considerations that are without importance. The etiology and pathology of most of these are as yet imperfectly known, especially in veterinary medicine. Among the numerous anomalies of the blood and nutrition we will describe: 1, anemia; 2, pernicious anemia; 3, hydremia; 4, watery cachexia of the sheep; 5, leukemia; 6, hemophilia; 7, scorbutus; 8, gout; 9, diabetes mellitus; 10, diabetes insipidus; 11, obesity; and, as addenda, 12, scrofulosis, and, 13, cancerous cachexia.

ANEMIA: CHLOROSIS.

Definition. *Anemia* is a morbid state characterized by a diminution of the blood mass and of its different elements: red corpuscles, leucocytes, hemoglobin, albuminoids, salts, and water of the plasma. In chlorosis the fundamental alteration is a decrease in the proportion of the hemoglobin of the blood; the quantity of this latter and the proportion of the other elements which enter into its composition may be normal. It is very frequent in the human species among young girls who have reached the age of puberty; chlorosis has not been the subject of any authentic observation in domestic animals. In veterinary medicine the word *chlorosis* has often been wrongly used as a synonym of *anemia*. No analogy can be established between chlorosis of man and that of animals.

Etiology. In a large number of cases anemia is due to heredity. It is to this influence that must be related the numerous cases which are observed in very young animals (dogs, cats, young pigs, colts,

calves). In the canine species much improved and delicate breeds and in the bovine species precocious breeds seem to be predisposed to it. Anemia may be caused by excessive loss of blood or by repeated hemorrhages. It is sometimes recognized in very fat animals, and in certain countries it is usual to precede the fattening process with successive bleedings. A poor or insufficient alimentation is another frequent cause of the disease (anemia of inanition). *Secondary or symptomatic anemia* occurs after diseases of long duration, exhausting work, parturition, great losses occasioned by the abnormal secretions. Progressive pernicious anemia will be described in the following chapter. Anemia of the sheep will also be the subject of a special study.

Symptoms. The skin and the mucous membrane are pale, *washed*, the color of milk. The animals, which are very weak, become tired after the slightest exertion; the circulation and respiration are accelerated; the pulse is slight and the temperature above the normal; the heart-sounds are sometimes obscured by pathological bruits; the appetite is capricious, the digestion is troubled, the muscles lose their tenacity, and the members often become oedematous. In newborn animals a rapid exhaustion occurs, which often leads to death within a short time. But in adult subjects anemia has usually a chronic course, and ends in recovery; in the sheep it is frequently complicated by hyperemia. Pernicious anemia is a very serious form.

Pathological anatomy. The blood is pale, "similar to bouillon," slightly or not at all coagulable. All the viscera are almost entirely bloodless. The dimensions of the heart and large bloodvessels are lessened (hyperplasia). When the disease is of long standing the principal organs (heart, liver, kidneys) undergo fatty degeneration.

Diagnosis. Paleness of the visible mucous membranes (buccal, vaginal, ocular) is a marked symptom. A quantitative analysis of the blood enables us to recognize a diminution of the proportion of hemoglobin. Zschokke recommends the use of the hemoglobino-meter of Gowers (colorimetric method). We are indebted to Schindelka for some interesting researches on the proportion of hemoglobin which is contained in the blood of the horse.

Treatment. Iron is the remedy *par excellence*. It is administered either by adding it to the food in a form which is easily assimilated or in subcutaneous injections. The simplest prepara-

tion is pulverized iron ; it is given, associated with sea-salt, for the horse and ox in a dose of 1 gramme daily, for the dog in a dose of 1 decigramme. In small animals we frequently use ferruginous tinctures, especially iron tincture with apple juice (*Tinctura ferri pomata*), diluted with a sufficient quantity of water ; we may prescribe for the dog :

Tinct. ferri malitis	5 grammes.
Distilled water	250 "

Two tablespoonfuls daily are to be given.

Polyemia or *plethora* is a morbid condition which is the opposite of anemia. It formerly occupied an important place in special treatises ; nowadays its existence is doubted. Through experiments upon animals Müller and Cohnheim have observed that an addition by transfusion of 50 to 80 per cent. of blood coming from subjects of the same species does not produce plethora. However, Bollinger¹ has shown that the proportion of blood varies much in the different animal species. While in the fat pig the blood mass is 2.2 per cent. of the body-weight, in the draft horse it is 13.5 per cent. Such variations force us to admit the possibility of the existence of plethora. This seems to be produced during the first period of fattening in young and strong animals which have worked till that period, and in females where the lacteal secretion has suddenly stopped. It is marked by a redness of the mucous membranes and a full and strong pulse ; it predisposes to congestion of the brain and lungs. In man plethora is specially due to irregularities of the regimen. It may be recognized at the autopsy. It is characterized by excess of blood, by hypertrophy of the heart, by amplification of the large bloodvessels and capillaries of the principal glands.

PERNICIOUS ANEMIA.

Etiology. Pernicious anemia, which is a malignant form of essential anemia, is a very serious disease, the etiology of which is but little known. Several peculiarities, however, indicate its infectious origin. Zschokke, who has seen it run its course in an enzootic state in a stable, has found in the patient's blood very fine bacilli, a fact which is confirmed by our personal observations. Excessive exertions, a prolonged stay in the stable, and previous

¹ Münch. medicin. Wochenschrift, 1886.

pneumonias seem to be predisposing causes of this anemia. Up to the present time about one dozen cases only have been mentioned in the horse. Imminger is said to have seen it in an enzootic state in the ox.

Serious *secondary* anemia may occur during the course of verminous diseases and long-continued suppuration, etc. Mégnin has observed it to some extent in cats and dogs which were affected by uncinariosis. (See vol. i., page 243.) But these symptomatic anemias do not deserve to be considered as special morbid states.

According to the researches made by Evans and Burke, the disease of the horse known in India under the name of *Surra* is a pernicious anemia. Evans states that it is produced by a filiform hematic protozoaire, which is characterized by very active movements.

Pathological anatomy. At the autopsy we are struck by the marked fluidity and small quantity of the blood. This liquid shows alterations altogether special, and which are wanting in ordinary essential anemia. The red corpuscles, which are very pale, have lost their regular characteristic form; according to the cases they may be found angular, elongated, club, biscuit, or drum-stick shaped; often near very small corpuscles (microcytes) there are others which are voluminous (macrocytes). It is to these morphological alterations of the red corpuscles that has been given the name of poikilocytoses. In the blood bacilli have several times been recognized (four to six on an average per field), and the length of which varied between a third and a half of the diameter of a normal red corpuscle.

Among other important alterations we must especially mention a degeneration of the myocardium, of the bloodvessels, of the muscles which control the phenomena of life, of the renal and hepatic cells, the existence of extensive hemorrhages in most of the organs, especially in the muscles, the large glands, and in the serous membranes, tumefaction of the liver, of the spleen, cellular infiltration of the bony marrow, and infarcts of hemoglobin in the kidneys. But none of the viscera have similar lesions.

Symptoms. This disease begins, as a rule, with symptoms of increasing weakness, and then its initial stage passes easily unnoticed; in some cases it is announced by a catarrhal process of the respiratory organs (cough, nasal discharge); in some others the symptoms of intense fever attract the attention. Sometimes the

mucous membranes are at first yellow-colored, but later they become more and more pale. Though the appetite is often preserved and the general conditions satisfactory, even at a quite advanced period of the disease, the animals are very soft and perspire on the slightest exertion. We invariably notice an intense irregular hyperemia, which shows remissions of several days and resists all antipyretics; during febrile attacks the pulse is considerably accelerated and the heart-sounds are marked by abnormal bruits. The examination of the patients does not reveal any organic disease. By means of the microscope we may recognize alterations of the blood (poikilocytosis, existence of macrocytes and microcytes).

Later, emaciation takes place, dropsies appear, and exhaustion becomes gradually marked; finally the animals die within a time which varies from a few months to several years. This termination is by far the most frequent. Death is always preceded by an intense pyretic attack, which is of long duration.

Differential diagnosis. A microscopic examination of the blood enables us to establish a diagnosis. It must be performed rapidly, for exposure of the blood to the air, or the addition of certain liquids, even of distilled water, may produce alterations similar to those of pernicious anemia. This examination is very apt to mislead the practitioner; study of the blood has almost always weakened the diagnosis, a circumstance which is as strange as it is far from the truth. The absence of a primary organic lesion, the profound auemia, and the intermittent fever, which is obstinate, and the cause of which cannot be found, are also elements which clear the diagnosis and ought to guide us. We distinguish this anemia from leukemia by the absence of the symptoms of this latter: a considerable increase in the number of white corpuscles, tumefaction of the lymphatic ganglions, and leukemic infarct. Hemorrhages are common to both these diseases.

Treatment. Preparations of iron constitute the most powerful regenerators of the blood; but their efficiency is much more uncertain than in common anemia. In the adult horse we give pulverized iron (in a dose of 2 to 5 grammes, associated with chloride of sodium and powdered aromatic flowers) or sulphate of iron (in a dose of 2 to 5 grammes, mixed with small quantities of carbonate of potash and aromatic powders). We may also try arsenious acid or Fowler's solution. This is administered in a dose of 10 grammes per day, which is gradually increased to 50 grammes. The fever

has been combated by the many antithermics: antipyrine, quinine, thalline, antifebrine, phenacetine, etc.; but in pernicious anemia the action of these agents is very weak. During the entire treatment the animals must be allowed to rest.

Pernicious anemia of man was first studied by Biermer in 1868. Its symptoms and its alterations are the same as those which were described by Zschokke in the horse. Its causes are imperfectly known. It is generally admitted that it is of an infectious nature. In the workingmen who were employed in the Saint Gothard tunnel, Perroncito observed a secondary pernicious anemia of parasitic origin, which was produced by ankylostomes. The same disease has been recognized in workingmen who were employed in mines and brickyards. Bothriocephalis may also determine a secondary pernicious anemia.

Quite recently Ponfick has undertaken experimental researches upon the pathology of pernicious anemia.¹ This experimenter has succeeded in producing the disease experimentally in animals by means of repeated intravenous injections of blood dissolvants (glycerin, pyrogallie acid, solutions of hemoglobin). It seems therefore to consist essentially of a chronic hemoglobinemia; the alterations of the red corpuscles would be the result of setting free the coloring-matter of the blood. In the support of this opinion we may refer to the presence in the kidney of infarct of hemoglobin, an alteration which we have recognized in the horse. During the course of chronic hemoglobinemia troubles of hematopœsis occur; oxygen is wanting, the organs undergo fatty degeneration, and the bloodvessels become ruptured. The exciting cause of hemoglobinemia occurring in pernicious anemia is unknown. It is possible that it is of microbic origin.

ADDENDUM.

Under the name of *scalma* Dieckerhoff² has described a new disease, of which he has gathered a series of observations in the horse, and which are characterized by the following symptoms: coughing, bilateral discharge, dyspnœa, fever, weakness, paleness of the mucous membranes; percussion and auscultation do not

¹ Silbermann: Berliner klinische Wochenschr., 1886.

² Dieckerhoff: Adam's Wochenschr., 1885; u. Lehrbuch der spec. Path. u. Therap., 1885.

reveal anything abnormal; the appetite is preserved; in certain cases there is an inflammation of the pharyngeal mucous membrane, and in some others a diffuse exudative pleurisy. The course is generally regular and the termination favorable. The period of convalescence lasts on an average from three to four weeks. The disease is of an endemic character, and may exist for two or three months or longer in the same stable. Dieckerhoff thinks that it must belong to the nosological group formerly known under the name of influenza.

We cannot possibly consider *scalma* as a clinical entity. Part of the cases observed by Dieckerhoff seem to be related to the morbid condition which we have just described. An enzootic appearance of the disease, a pronounced paleness of the mucous membranes, the weakness of the patients, the irregular course of the fever, preservation of the appetite, the absence of any localization with the exception of that which is produced upon the respiratory mucous membrane, a long convalescence: all these symptoms are observed in pernicious anemia. Although this disease usually ends in death, we have, with Zschokke, seen less serious cases. On the other hand, we have observed cases of pharyngeal angina and contagious pneumonia which offered a great analogy to some cases of *scalma* which have been mentioned by Dieckerhoff.

HYDREMIA: DROPSY IN GENERAL.

General considerations. General pathology distinguishes three forms of dropsy: 1, *mechanical dropsy*, which is determined by lesions that obstruct the return circulation (heart, lung, kidney, and liver diseases); 2, *inflammatory dropsy*; 3, *hydremic dropsy*, which is produced by excessive fluidity of the blood and by alterations of the vascular walls. This latter alone constitutes an essential primary disease. General dropsy is mostly found in the sheep as an epiphenomenon of distomiasis; but in this species and in the pig it is sometimes developed during the course of auemia without the intervention of any parasite (see the following chapter). The disease of draught oxen observed in refineries and described under the name of *general dropsy of the connective tissue*, a disease which is also recognized in horses used in these factories (Garcin), is particularly interesting from a clinical standpoint.

Etiology. The causes of hydremic dropsy are similar to those

of anemia ; a hereditary predisposition and impaired nutrition constitute its most important factors. In sugar factories the disease is produced by the consumption of the watery refuse of turnips. On account of the modern process of diffusion the turnip waste, which contains as much as 95 per cent. of water, is very poor in protein or nitrogenous matter ($\frac{1}{2}$ per cent.). An alimentation which is composed exclusively of this watery and little nutritive substance, and the excessive work which is exacted from the animals, end by producing hydremia. The rarity of this disease in cows is due to the little use which is made of them as working animals and to the abundance of the lacteal secretion, by which means they get rid of the excess of ingested liquid.

Pathological anatomy. The blood is watery and but little colored ; it coagulates slowly. All the organs as well as the subcutaneous connective tissue sometimes contain a clear, serous, uncoagulated, uncolored, or yellowish liquid, which is poor in albumin, and which holds in suspension endothelial cells and leucocytes, in other instances an inflammatory exudate.

At the autopsy of draught oxen which had died of hydremia at sugar factories we are at first struck by the absence of rigidity of the cadaver and by the paleness and flaccidity of all the muscles. The subcutaneous and inter-muscular connective tissue is infiltrated with serum ; in the splanchnic cavities we find more or less abundant exudates ; the intestines are contracted, pale, and deprived of fat ; the brain is œdematous ; generally we recognize also lesions of chronic intestinal catarrh.

Symptoms. According to the localization of the discharge we distinguish dropsy of the skin (anasarca), of the abdomen (ascites), of the thorax (hydrothorax), of the pericardium (hydro-pericarditis). Subcutaneous dropsy is at first observed in the depending regions (legs, abdomen, chest, testicles) ; in the sheep it appears upon the head—a phenomenon which is due to the low position of this organ when the animals are at pasture ; the skin, which is tumefied and of doughy consistency, retains the imprint of the finger, but it is neither hot nor painful. The patients become more and more weak ; dyspnoëic phenomena and digestive troubles occur ; the mucous membranes, which are always pale, are sometimes the seat of an œdematous tumefaction ; the pulse is scarcely perceptible.

The disease of oxen which are fed upon sugar factory refuse has a slow evolution. In the beginning we observe depression and

weakness, also a paleness of the mucous membranes; though the appetite remains excellent, the general condition becomes more and more altered, the hair is dull and bristly; the animals pass an abundant quantity of clear watery urine. Later, digestive troubles occur, which are especially marked by alternations of constipation and fetid diarrhea; there is often ptyalism. Dropsy appears in the extremities, which become enlarged and render the walk heavy and painful. Soon œdematous tumefactions are developed in the abdomen, fetlock, and chest; exudations are produced in the large splanchnic cavities, especially in the abdominal cavity, the dimensions of which increase. Finally, a time arrives when the exhausted animals can no longer preserve a standing position, and they remain constantly stretched upon the ground. Death takes place in from three to six months, except when treatment is commenced from the beginning of the disease.

Treatment. We must, before all, attack the cause of the disease—that is to say, change the regimen when the trouble is observed in oxen which are kept in sugar factories. Symptomatic treatment consists of making use of diuretics and purgatives. (See Treatment of Ascites and of Hydrothorax.)

CHRONIC ANEMIA AND DROPSY OF SHEEP AND OXEN.

Chlorosis: Watery Cachexia.

OBSERVATIONS ON THE EXPRESSION OF “ROT.” The term “rot,” used in the early period of veterinary medicine, has been perpetuated to this day. Gerlach, in his *Treatise on Legal Veterinary Medicine*, has pointed out the want of precision of this word, but his criticism remains unheeded. This is undoubtedly due to the fact that in certain countries legislation concerning such diseases as set aside a contract of sale still includes “sheep rot.” This appellation might serve to designate any disease which is accompanied by emaciation, weakness, anemia, or dropsy. Formerly it was also applied to tuberculosis of the ox. Some modern authors have applied it exclusively to distomiasis, others to tæniasis, and others to chronic anemia (chlorosis) and to non-parasitic idiopathic dropsy. Distomiasis and tæniasis having been described (see vol. i., pages 220–263), we have to study here a non-para-

sitic constitutional enzootic disease, the principal manifestations of which are chronic anemia and general dropsy. It is generally observed in the sheep and ox (according to Spinola, it is also found in the pig). In order to conform to custom, it may be designated under the name of *watery cachexia*, which is much more significant than the primitive and empiric expression of "rot."

Etiology. The causes of chronic anemia and dropsy of the sheep and ox may be grouped in two classes. The first comprises badly-composed rations (insufficient nutritive principles), swampy, sandy, or turfy pastures, and, especially in the ox, a very watery alimentation (slops, tainted fodder) and a defective hygiene (badly-constructed and badly-kept stables). In the other we consider the inclemency of the season (cold and damp weather, being on pastures exposed to cold winds, also pens or folds situated on damp and cold ground). The disease is often observed after rainy years or after freshets; under these circumstances it may occasion considerable losses.

Pathological anatomy. The blood shows constant alterations; it is not abundant, and is of a pale color and "similar to bouillon;" the subcutaneous connective tissue of the neck and shoulders, abdomen, and chest is the seat of an œdematous infiltration. The inter-muscular connective tissue is also infiltrated and gelatinous; the muscles are flabby and discolored; the abdominal viscera are atrophied and pale; the lungs are whitish, inflated-like; the heart is flabby and soft. In the splanchnic cavities and in the cerebral ventricles, between the meninges and in the rachidian canal, we find a transparent slightly-yellowish liquid.

Symptoms. In some cases the appetite remains normal for quite a long time, but nevertheless the animals become weak, the gait is dragging and staggering, debility and emaciation become rapidly accentuated; in others there is inappetence from the start. The mucous membranes become pale; soon they seem altogether bloodless. The conjunctiva is frequently the seat of an œdematous tumefaction, the sclerotic shows a bluish coloration; it is not rare to observe in the eyes a mucous discharge. The skin, which is pale and infiltrated, has "the appearance of suet;" the wool is dull, dry, and falls, or may be pulled out with the greatest ease; the circulation is accelerated. In the ox the hair is bristly and the epidermic desquamation produces an abundant furfur. Œdematous tumefactions are developed in the intermaxillary space, on

the neck and shoulders, under the chest and the abdomen; we recognize all the symptoms of ascites. Later an abundant colliquative diarrhea occurs. The patients, which are exhausted, are too weak even to move, and remain constantly down; generally they die within a few months, at the latest within a year.

Treatment. We must modify the conditions under which they are kept and also the alimentation; this is the only efficient indication. During the first stage of the disease it is possible to save the animals when we are able to give them a nutritious food which is rich in nitrogenized matter (grain and good hay). The most favorable medicinal agents are preparations of iron, chloride of sodium, lime, and aromatic stomachics: gentian, sweet flag, absinthe, juniper berries, etc. For a flock of 100 sheep we may prescribe: chloride of sodium, 500 grammes; powdered gentian, powdered rhizom d'acore (sweet flag), 250 grammes; powdered sulphate of iron, 100 grammes; to be made into a mass which the animals can lick, or given in association with barley malt.

LEUKEMIA.

NATURE. Leukemia is an anomaly of the composition of the blood which is characterized by a considerable and permanent increase of the white corpuscles. In a normal state the proportion of the white to the red corpuscles is 1 to 350. In leukemia we find 1 white corpuscle for 50, 20, 10, or even 2 red.¹ According to its starting-point, we recognize therein three forms, which may be combined in several ways:

1. A *lineal* form, in which the primary and principal lesion is hyperplasia of the spleen. It is distinguished from the other two by the existence of large-sized and multinuclear leucocytes.

2. A *lymphatic* form, the predominating alteration of which is hyperplasia of the lymphatic ganglions.

3. A *myelogenic* form, which has its origin in the hyperplastic phenomena of the red marrow of the bones.

Leukemia is not so rare in domestic animals as is generally be-

¹ The adult and healthy man possesses on an average five millions of red corpuscles per cubic millimetre, and nearly all warm-blooded animals present the same globular richness. (Hayem: *Du Sang et des Altérations Anatomiques*. Paris, 1889.) In the adult horse and in the dog the average number of red corpuscles is 7,500,000, and the proportion of leucocytes is 1 to 800-1100, instead of 1 to 350 as in man (Nocard).

lieved; the facts which are mentioned in our different publications confirm this statement. It has been observed in the horse, ox, dog, pig, and the cat (Eberth has also recognized it in the mouse). No case has as yet been mentioned in the sheep or goat.

Its causes are also less known in domestic animals than in man. In the human species debilitating influences: traumatisms, intermittent fever, syphilis, etc., are considered as causes. The rôle which these latter diseases seem to play in the genesis of leukemia has led us to consider this as an infectious disease, the primary lesion of which involves one of the blood-making organs: spleen, lymphatic ganglions, red marrow of the bones.

Pathological anatomy. The blood, which is discolored and very pale, no longer holds in suspension the corpuscles which are found in its mass (white blood). It coagulates slowly, separating a grayish puriform matter which forms a layer between the red and white clot. The blood which is contained in the heart and large bloodvessels is of slight consistency, viscous, and composed in major part of leucocytes. On account of the diminution in the number of red corpuscles the specific gravity of the blood is lowered (from 1055 it decreases to 1040); this liquid contains numerous abnormal products: formic, acetic, lactic, and uric acids; xanthine, hypoxanthine, leucine, etc.

The white corpuscles show all the morphologic varieties. Some have preserved their normal structure and are provided with a nucleus; most of these are homogeneous, transparent (embryonal type); others, that are colored by hemoglobin, contain a nucleus and fatty corpuscles. Between these principal types we find all possible transitory states.

In the *lineal* form the spleen is often doubled or trebled in size; in a case which was observed in the horse by Leisering it weighed 28 kilogrammes (normal weight 500 to 760 grammes). Johne found a spleen of a pig weighing 2 kilogrammes 400 grammes (normal weight 150 grammes). The edges of this organ are rounded, its consistency is firm, its follicles sometimes reach the dimensions of a pea; but the histological structure of the splenic tissue is kept normal (simple hyperplasia).

In *lymphatic* leukemia the lymphatic ganglions are hypertrophied and may form large tumors, the consistency of which is soft. Generally all the ganglions of the head, of the neck and shoulders, of the extremities, and of the abdominal and pectoral cavities are tume-

fied and hypertrophied. Peyer's plaques and the solitary follicles of the intestine participate but little or not at all in the process and the hypertrophy.

In the marrow of the bone we frequently recognize alterations of diffuse hyperplasia and a cellular infiltration. In the *pyoid* form of myelogenous leukemia the marrow, which is extremely rich in white corpuscles, shows a puriform aspect. In the *lymphadenoid* form it has the consistency of raspberry jelly.

Besides these lesions we find leukemic infarct and lymphoid tumors around the bloodvessels of the liver, spleen, kidneys, uterus, bladder, lungs, subcutaneous connective tissue, the serous membranes, mucous membranes, retina, etc. Leukemic infarcts consist of a diffuse infiltration of the tissue by white corpuscles, an infiltration which surrounds the bloodvessels with a sort of whitish-gray membrane. The lymphoid neoplasms are circumscribed tumors, the histological constitution of which is that of lymphatic ganglions. The hemorrhages which occur at times during the course of leukemia may have left some traces in several organs (mucous membranes, kidneys, etc.).

Symptoms. The beginning of leukemia is insidious and its symptoms vague. There is a more or less marked weakness, fatigue on the slightest exertion, dyspnoea, abundant sudations after the least exertion, vertiginous attacks, a remarkable paleness of the mucous membranes and skin; such are usually its first manifestations. As a general rule, the circulation is accelerated and the pulse frequent, small, and irregular. In some cases auscultation of the heart enables us to perceive abnormal bruits of anemia. There are patients in which all the superficial lymphatic ganglions are symmetrically hypertrophied. Nocard has observed a case of this kind in a cow. In others hypertrophy is remarked in a few ganglions only, but especially in the peri-pharyngeal. In the horse we may sometimes recognize hypertrophy of the spleen, which forms a more or less prominent tumefaction in the left flank. The appetite remains normal for a long time, later it becomes capricious and gastric troubles appear (diarrhea, etc.). At an advanced period of the disease we often observe hemorrhages on several mucous membranes (nasal cavities, intestine, bladder) and diffuse œdematous tumefactions. We do not recognize any organic disease to which this *ensemble* of symptoms may be ascribed. A diagnosis can only be established by a microscopical examination of the blood; this

may be obtained directly from a vein, or in the skin by means of scarification made upon that membrane, and it is to be examined without subjecting it to any preparation.

Leukemia is a disease with a very serious prognosis; its course is always chronic; it is prolonged for months and even for years. Very often it is only recognized at the autopsy.

Differential diagnosis. If the microscope enables us easily to distinguish leukemia from other diseases of the blood, and particularly from pernicious anemia, the existence of white corpuscles in greater proportion is by no means sufficient in all cases to prove the presence of this condition. A considerable increase in the number of leucocytes is in fact observed normally under several circumstances: at the time of gestation, immediately after meals, after bleeding, and during the course of inflammatory diseases in general; this phenomenon is designated under the name of *leucocytosis*. In glanders, as in some cases of leukemia, we may observe a slight leucocytosis and also hemorrhages from different mucous membranes. We ought only to diagnose leukemia if the permanent increase of white corpuscles reaches the proportions which have been heretofore indicated.

Treatment offers little chance of success. The regimen must be regulated, and preparations of iron administered. Arsenious acid may be tried. In man quinine is particularly recommended.

Under the name of *pseudo-leukemia* (malignant lympho-sarcoma) a chronic constitutional disease has been mentioned by authors which consists of a general hyperplasia of the lymphatic ganglions without any increase in the number of white corpuscles. Lustig and Dieckerhoff have observed it in the horse, and Fröhner in the dog. Its pathology is unknown, as well as the relations which may exist between it and other constitutional diseases. But a transformation of pseudo-leukemia into true leukemia has been several times recognized.

In the beginning ganglionic hyperplasia is the only important symptom; no functional trouble exists; later, the clinical picture of serious anemia is outlined and gradually accentuated.

HEMOPHILIA.

NATURE. Hemophilia is an hereditary hemorrhagic diathesis, which is marked by a tendency to abundant spontaneous hemorrhages, or by a considerable loss of blood as a consequence of the

most insignificant traumatisms, which may also endanger life. It is to be remarked that the spontaneous hemorrhages observed in hemophilias are generally related to previous traumatisms. The usual determining causes of hemorrhages are superficial wounds of the skin, incision of the fistulous tracts, dilatation of the wounds of castration (Siedamgrotzky's case and our personal observations), application of setons or of trochisci (Köhne, Dieckerhoff), castration, ulcers accompanying leg grease (Köhne), etc. In veterinary medicine the disease has been found exclusively in the horse.

In opposition to hemophilia as a morbid condition we have the secondary hemorrhage which is observed in some septic or toxic diseases (septicemia and petechial fever), as well as in different anomalies of the blood (leukemia, pernicious anemia, etc.).

We know little about the causes of hemophilia. In man we have several times observed an abnormal narrowing of the large bloodvessels and a thinning of the terminal vessels—that is to say, a hypoplasia of the vascular system. In the human species the disease is often transmitted by heredity (hemophilia of families).

Quite recently Dieckerhoff has considered hemophilia as a disease of a toxic nature. But that is a hypothesis which does not seem to us to be well founded. If, in fact, hemophilia was due to a poisoning, the hemorrhages would be produced at the same time in several regions, as is the case in petechial fever, and not from one point only; neither would its long duration and its obstinate character be explained. In petechial fever and septicemia hemorrhages are not ordinarily dangerous, and cause death only exceptionally. In fine, Dieckerhoff's opinion has also against it an hereditary transmission of hemophilia in the human species. We cannot understand this transmission if we admit the toxic nature of the disease.

Symptoms. Hemophilic animals usually present nothing abnormal directly preceding the hemorrhage. Catarrhs of the respiratory and digestive mucous membranes, which are looked upon by Dieckerhoff as prodromes of hemophilia, have, in fact, no relation to this latter. Though the contrary is indeed the case, the disease seems latent till a wound is inflicted; then the blood escapes sheet-like from an insignificant wound, and, notwithstanding the use of astringents, the hemorrhage persists for hours, sometimes for whole days. Soon the blood is watery and incoagulable, the mucous membranes are pale, and the pulse rapid. The patients sink gradually; in some cases death occurs under the eyes of the

veterinarian, who is powerless to stop the hemorrhage; more rarely hemostasis is obtained within a few hours, and the bloody discharge may reappear from one moment to another. The prognosis is always very serious. In domestic animals we have never observed spontaneous hemorrhages during the course of hemophilia.

Treatment. Though styptics are of little efficacy, they have nevertheless given some service; it is therefore advisable to resort to them. After having compressed the wound by means of a tampon or ligature we must employ hemostatics, perchloride of iron, tannin, alum, cresol, or effective cauterization. Internally we may also administer some of these agents, and more especially ergot of rye and acetate of lead. [Direct transfusion of blood has in a large number of recorded cases proved very beneficial by supplying the blood of the diseased animal with the elements necessary to produce coagulation in the open capillary vessels. (Fibrin Hematitis, etc.).—W. L. Z.]

SCORBUTUS (Scurvy).

GENERAL CONSIDERATIONS UPON THE EXPRESSION "SCORBUTUS." Scurvy, which is, as a rule, an epidemic disease, was formerly frequent among ships' crews and the inhabitants of besieged cities; nowadays it is met with in prisons and barracks; isolated cases are sometimes observed. Scurvy is a hemorrhagic disease; it is announced by certain general symptoms (weakness, anemia, rheumatismal pains), and is marked by cutaneous hemorrhages, cyanosis, tumefaction of the gums, which rapidly begin to bleed, and by a consecutive ulcerous stomatitis; it occasions, besides, subcutaneous, intermuscular, mucous, and visceral hemorrhages; finally, it is accompanied by several complications (pneumonia, pleurisy, arthritis). It is probably of an infectious nature. The disease was formerly attributed to defective dwellings, unfavorable climate, and a poor quality of food, which was unsuitable or not sufficiently varied (salt meat and deprivation of vegetables during long sea-voyages). These seem to act only as predisposing conditions. Garrod's theory, in which scurvy was ascribed to the want or insufficiency of potash salts in food, has been abandoned.

In veterinary medicine scurvy has been recognized in the dog and pig. In the latter it is known under the vulgar name of "hair rot." In the other species its existence is doubtful. For-

merly the name of scurvy was given to any hemorrhage which took place from the gums; thus, most instances which have been mentioned as occurring in the dog refer either to common ulcerative stomatitis or to poisoning by mercury, lead, or phosphorus. The symptomatology of cases of *pseudo-scorbutus* observed in the horse is that of petechial fever and septic processes. In the sheep and goat cases of anemia and rhachitis without doubt have been mistaken for scurvy. Besides, most of the descriptions which have been given to it by authors are inexact. The question of knowing whether scurvy exists in the horse, sheep, and other domestic animals, the dog and pig excepted, remains thus to be solved. Even in the canine species we have found it but very rarely.

Etiology. In the pig the alimentary regimen and defective feeding and stabling, damp pens that are badly ventilated, and lack of healthy exercise have been considered as the first and chief causes; but these influences act, without doubt, only as predisposing causes. In this species swine plague has also been considered as a cause of scurvy (Cornevin, Hess). Its pathology is far from being clear. It is generally admitted that it is of infectious origin. These etiological considerations are also applied to the dog. Of all animals it is the most exposed to the pathologic influences which generate scurvy in man.

Symptoms. The disease begins with weakness and disturbance of the appetite. The gums assume a violet coloration; they soften and bleed with the greatest ease. The teeth become loose and fall out; there is ptyalism present; the mouth exhales an infectious odor. At the same time the hairs are detached from their bulbs; they may be pulled out with the slightest effort, or are spontaneously detached from the bulb; the root is bloody (hair rot). In the dog and pig we remark upon the surface of the skin reddish-blue spots or tracks (ecchymoses, vibices); in the pig these hemorrhages may be followed by deep ulcerations of the integument. In some cases we have recognized articular tumefactions. In the dog, independently of gingival hemorrhages, we observe epistaxis, retinal hemorrhages which are visible on ophthalmoscopic examination, and sometimes intestinal hemorrhages. Emaciation and weakness become gradually marked, diarrhea is permanent, and when the disease is left to itself the animals die through increasing exhaustion.

Pathological anatomy. At the autopsy we find numerous

hemorrhages in the skin and subcutaneous connective tissue, in the mucous membranes, under the serous membranes, in the brain, etc. The blood is black and fluid, or hardly coagulated.

Treatment. Two principal indications are to be fulfilled: the alimentary regimen must be changed, and the hygienic conditions to which the animals are subjected should be improved. It is also proper to administer bitters, astringents (gentian, quinquina, quinine), and preparations of iron. It is customary to give the patients fruit, acorns, or chestnuts.

The disease which is described under the name of SCORBUTUS OF THE SHEEP coexists frequently with watery cachexia and distomiasis (Lowak). In this disease the teeth become loose and fall out, the gums bleed easily, and finally ulcerate; there is ptyalism, and the animals exhale a fetid odor. Some veterinarians have recognized in the mouth bony ulcerations, which gradually enlarge and finally perforate the palate and reach the bones of the face, which are softened as in osteoporosis.

Gips, who has made a special study of this disease, thinks that it has nothing in common with true scurvy; according to him, it must be considered as a pernicious stomatitis, produced by wounds of the gums, which have but little resistance, and also by the penetration into the dental alveoli of alimentary matters, fungi, and microbes. It is observed exclusively in anemic animals and in certain breeds, also in lambs which are kept permanently stabled. As soon as pasture feeding is stopped it appears in weak animals of fine wool-producing breeds, and assumes at once an enzootic character. Lambs of German domestic breeds, of English meat-producing breeds, as well as adult animals, whatever their origin, are not affected by it. In the spring, when the animals return to pasture, the disease disappears of itself. It is particularly fatal after damp summers and when the sheep are fed on watery substances (residues, potatoes, etc.).

These are, according to Gips, its principal symptoms: the animals are weak, the appetite is diminished or capricious, the mouth foamy; the gums are red and tumefied; the teeth become loosened successively, first, upon the corners and then on other incisors and molars; the epithelium is destroyed over large surfaces. Alveolar periostitis, which occurs later, produces a falling out of the teeth; alimentary particles become lodged in the alveoli; the maxillæ become tumefied, then partially necrosed; prehension and

mastication are laborious and painful. At this period anemia, weakness, and emaciation make rapid progress; the patients finally die in a cachectic condition. At the autopsy we find alterations of advanced anemia; the blood is fluid, clear, and will scarcely color white paper; the fat has undergone a gelatinous retrogression. We detect also lesions of general dropsy and chronic intestinal catarrh.

From a therapeutic standpoint Gips recommends giving the patients food which contains much nitrogen and is easy of digestion (hay, oats, barley, peas, vetches, lupine), and to drive them as soon as possible to pasture, to stable the animals in clean, spacious, well-ventilated sheepfolds, which are kept at a temperature of 8° to 10° R. Sulphate of iron dissolved in drinking-water (0.1 gramme per animal per day) is beneficial. Phosphate of lime possesses no efficiency. The gums must be treated with astringent or antiseptic solutions.

GOUT: URIC ARTHRITIS.

NATURE. Gout is a morbid condition, the essential character of which is an increase in the proportion of uric acid contained in the blood. This acid and the urates are deposited in certain articulations (uric arthritis) or in the track of the organs (visceral gout). It has been observed in the chicken, pigeon, goose, turkey, ostrich, etc. Old animals seem to be predisposed to it. Its existence in the bird species is without doubt due to the large amount of uric acid contained in the excrements of birds; in some cases the elimination of this acid appears to be obstructed. In the gallinaceous species it is possible to produce uric arthritis experimentally by means of a ligature of the ureters (Ebstein). The predominance in the blood of substances which are of acid reaction favors the precipitation of uric acid. But we do not possess any other precise fact bearing upon the etiology of gout. In man authors attribute the disease to heredity, to eating food rich in nitrogen, cold, etc. It is not known whether the accumulation of uric acid in the blood is due to increase in its production or to defective elimination. In the tissue this acid produces necrosis by its escharotic action, then by degrees it becomes crystallized therein (Ebstein).

Symptoms. The articulations most commonly and most seriously affected in chickens are those of the metatarsal phalanges and

tarsus; later we have those of the metacarpus, carpus, and elbow. In the beginning we observe on these joints a diffuse, soft, and painful tumefaction, which develops by degrees. Frequently the inner fascia of the metatarsal articulation shows knotty tumors, which are from the size of a pea to that of a hazelnut; they are hard at certain points and fluctuating at others; they are generally hot, painful, and surrounded by a red zone. The hypertrophied epidermis which covers the gouty tumors becomes exfoliated in thick scales; these lesions are very frequently opened spontaneously and permit the escape of a granular, yellowish-gray matter, similar to talc, which is a steatite formed principally of uric acid crystals, urates of ammonia, and lime; ulcers with bleeding edges and a grayish centre are formed and extend deeply as far as the bony tissue; the epiphyses become necrosed or ankylosed, the phalanges are thickened and incurved. Sometimes similar tumors are developed along the tendons; to the touch they give a sensation of concreted masses.

The general condition is usually affected. The walk is difficult; the suffering member no longer lends support; the animals avoid the slightest movements. Emaciation, weakness, and anemia occur; the erectile organs become pale and an abundant diarrhea appears. In serious cases the patients die in a short time.

Visceral gout is only recognized at the autopsy. It is marked by calcareous tubercles, by incrustations in the serous membranes, the vascular walls, pericardium, intestine, liver, spleen, and the subcutaneous connective tissue.

Diagnosis. Gouty arthritis may easily be confounded with the purulent and caseous forms of tuberculous arthritis, which are very frequent in domestic gallinaceæ. The diagnosis is insured by a microscopic or chemical recognition of the urates. In a microscopic examination the sandy concretions appear to be constituted of a true entanglement of very fine needles; the most consistent parts are also composed of crystals. A chemical demonstration of the urates is made by means of nitric acid and ammonia. We add to the concretions a few drops of nitric acid, then we evaporate to dryness in a watch-glass at a moderate temperature; a red onion-like mass is formed, which takes a beautiful reddish-purple color by the addition of a drop of ammonia; with a few drops of caustic potash we obtain a bluish-purple coloration. Gouty arthritis is remarkable for its slow course.

Treatment. The treatment of gout in birds is principally surgical. It consists in puncturing the affected articulations. Internally we may administer alkalies in the drinking-water. We prescribe, as a rule, artificial Carlsbad salts in a daily dose of a small pinch in the drinking-water. In man this salt is also used associated with other alkalies and given in drinking-water. It acts as a solvent of the urates.

DIABETES MELLITUS.

GENERALITIES ON THE NATURE OF DIABETES MELLITUS. Diabetes mellitus is a morbid condition shown by the presence of a considerable amount of sugar in the urine. A passing elimination of grape sugar by the renal organs is designated under the name of *glycosuria* or *melituria*. In the ox and the sheep the urine contains, when normal, very small quantities of sugar; in nursing female dogs at the time of weaning it also contains a small proportion of it; we ought to add that, in this latter case, we do not refer to grape sugar, but to sugar of milk (*lactosuria*). At the present time the nature of diabetes mellitus is unknown. None of the numerous theories which have been formulated on the subject of the pathology of the diabetes of man has been proven. In many cases this disease, like albuminuria, is probably but a symptom of very different affections. It is due to a nutritive trouble under the influence of which sugar leaves the blood without having undergone its usual metamorphoses. The expression diabetes mellitus signifies, therefore, a *permanent augmentation of sugar in the blood*. The source of the sugar appears to be, on the one hand, in the carbohydrates which are ingested with the food, and, on the other hand, by a more active destruction of the albumin, as indicated by the increase of the proportion of urea. In man, in a normal state, the proportion of sugar in the blood is 0.05 per cent.; in diabetes mellitus it may increase even to 1 per cent.

In veterinary medicine diabetes mellitus has been recognized several times in the dog; we have observed many cases ourselves. Three instances of it have been mentioned in the horse; two of these were reported by Heiss and the other by Roueff. U. Leblanc has found it once in the ape. Other clinical facts which have been published as examples of diabetes mellitus should not be accepted without reserve; they are very probably related to passing glyco-

suria. In the canine species females and old dogs are mostly affected; we have observed it twice in the male dog. Schmitt recognized it in a dog which had been swimming across the river Mein; in this case he ascribed it to the action of cold. It is possible that violent exertions or excessive work favor its development.

Melituria, or transitory glycosuria, may be owing to various causes. Claude Bernard has determined an experimental glycosuria in the rabbit by pricking the floor of the fourth ventricle. In man we have observed glycosuria after certain encephalic diseases (cerebro-spinal meningitis, cerebral commotion, fractures of the cranium, apoplexy, psychosis); also, in a large number of cases have authors related to these diseases diabetes mellitus which had occurred previously. According to Cyon, glycosuria would appear to be a consequence of lesions which involved the optic layers, the sympathetic, and the spinal cord.¹ It is recognized in the course of some acute infectious diseases and in a large number of poisonings—after the administration of morphine (a fact observed in the horse), chloroform, chloral, ether, alcohol, and nitrite of amyl, etc. We have seen it developed in cats which were kept in confinement in order to practise surgical operations on them (diabetes of constraint). Quite recently Mering has produced it by the administration of phloridzine (glycoside of the apple-tree root).

An experimental production of melituria by a wound of the medulla oblongata or of another region of the cerebro-spinal axis, and its appearance during the course of some diseases of the brain, or under the influence of encephalic poisons, have caused it to be considered as a disease which is especially connected with alterations of the central nervous system. It has been supposed that the latter's rôle in the genesis of the diabetes consisted of troubles of the organic mutation or in a vasomotor hyperemia of the liver, which activates the glycogenic function of this organ. But these hypotheses are not well founded, and the influence exerted by the cerebro-spinal centres in the pathology of diabetes is most obscure. We are therefore not much more advanced on the subject of the relations which exist between diabetes and glycogenesis. In some

¹ Extirpation of the pancreas produces an intense glycosuria which, in most cases, persists until death. (Von Mering and Minkowski: Diabetes mellitus nach total Pankreasextirpation, Strasburg, 1889, and Hédon: Archiv de médec. experiment. et de d'anatom. pathol., 1891.)—N. D. T.

cases the liver of diabetic animals has been found hypertrophied and congested (Wolff has recognized these alterations in the dog).

Symptoms. In the DOG the first symptoms of diabetes mellitus are depression, weakness, fatigue, and emaciation; generally hunger and thirst are intense; micturition is frequent and abundant; the specific gravity of the urine is increased; it varies between 1040 and 1060. The proportion of grape sugar contained in this liquid ranges from 7 to 12 per cent. (Wolff and we ourselves have found from 7 to 8 per cent.; Haltenhoff, 12 per cent.); there are cases where glycosuria may be recognized by the sweetish taste of the urine. Sometimes we see the development of a bilateral diabetic cataract, which leads in time to complete blindness (Wolff, Haltenhoff, personal observations). In a dog affected with experimental melituria, which had been produced by the ingestion of a considerable quantity of sugar, Schulz and Strübing observed in both eyes a loosening of the retina. We recognize also vomiting, coughing, diarrhea, and hemorrhages of the mucous membranes (Thiernesse), and ulcerations of the cornea. Toward the end of the disease weakness becomes extreme.

The course is always slow and chronic. The prognosis is serious even in cases where transitory improvement occurs. The disease is prolonged for months; it usually terminates in death.

In the HORSE, Heiss has observed diabetes mellitus in two animals which were of Belgian breed, ten and eleven years old, and matched. The first symptoms were weakness, depression, fatigue, nutritive troubles, emaciation, and dull hair. The patients ingested from three to five times more water than in a normal state. The appetite was preserved till the approach of death. The urine had a loathsome and disagreeable odor; with Trommer's test it gave a reddish-yellow precipitate largely composed of peroxide of copper; it contained on an average 3.75 per cent. of grape sugar. During the fifth week these two patients were affected with diabetic cataract and corneal ulcers. They died of marasmus within two months. At the autopsy we observed a peculiar yellowish coloration of the liver (clay-yellow) and a slight tumefaction of this organ.

Rueff has mentioned another case of diabetes mellitus which was observed in a ten-year old gelding. This animal, which had been sick for five weeks, was very weak and in poor condition. The appetite was preserved, the thirst intense, and the urinary

secretion considerably increased. The urine, which was examined by Werner, had a specific gravity of 1052, and contained 5.85 per cent. of sugar. The treatment employed did not produce any improvement.¹

During the course of diabetes mellitus in *man* we observe also furunculus, carbuncular cutaneous accidents, gangrenous alterations, which are produced by a permanent decubitus, impotence, excoriations upon the genital organs, phimosis, anhidrosis, neuralgias, diabetic coma (quite recently authors have attributed diabetic coma to a poisoning of the blood by aceto-acetic and oxybutyric acids; the urine frequently contains acetones); the expired air has a disagreeable odor. In about half of the cases diabetes is complicated with pulmonary phthisis.

Diagnosis. Weakness and emaciation progress, while the appetite and thirst are increased, the urinary secretion is very abundant, the presence of cataract should cause the practitioner to suspect diabetes mellitus. But a diagnosis may only be safely established by a chemical or physical examination of the urine for grape sugar. This is easily demonstrated by Trommer's test. We add to the urine potash lye, in order to give to it a distinctly alkaline reaction, then we add a solution of sulphate of copper to it, drop by drop, until the liquid shows a dark blue coloration (formation of hydrate of oxide of copper). On applying heat the dark blue color gradually becomes a dull yellow or reddish-yellow (formation of hydrate of peroxide of copper); a precipitate which is formed by this salt is deposited on the bottom of the glass when the urine contains more than 0.5 per cent. of grape sugar. A quantitative analysis by means of Fehling's liquor is based on the same reaction. Böttger's method is based upon a reduction of the subnitrate of bismuth into metallic bismuth, when we bring to a boil an alkaline diabetic urine and add a certain quantity of this salt. By heating

¹ Diabetes mellitus has been recognized by Darbas in an ox, in the province of Gascony, which was six years old. The animal had just been purchased, and the circumstances under which the disease made its appearance could not be determined. It was noticed that he was subject to frequent micturition, which was slight and not painful; the appetite was preserved, the conjunctiva had a rose tint, the skin was dry, and the hair dull. During work he would stop every five or six minutes and eject a small quantity of very clear, slightly amber-colored urine. Analysis of this liquid demonstrated the presence of a small quantity of sugar, the proportion of which was not determined. They tried in vain to fatten the patient; notwithstanding abundant food, he continued to lose flesh and had to be killed. (See *Revue Vét.*, 1890.)—N. D. T.

carefully the superficial layer of the diabetic urine, which has been rendered alkaline by means of potash lye, we obtain a dark brown coloration (Moore's test). The sugar of the urine may be recognized physically by means of polarization; it produces a deviation of the spectra to the right (from which the name dextrose is derived). With these instruments a quantitative analysis is very simple. As a rule, we use Mitschelich's apparatus.

In the fermentation test, which is much more complicated, we add to the urine a little lye, which transforms the sugar into alcohol and carbonic acid. The loss of weight resulting from the separation of this acid enables us to determine the amount of grape sugar in the urine.

Treatment. The principal indication in the treatment of diabetes mellitus is a reduction of the carbohydrates contained in the food. The most important factors of the formation of sugar are the amylaceæ, the sugar itself and gelatinous substances; in the ration they are to be replaced by albuminoids and fatty matter. In the dog it is advisable to give as much meat as possible; the feeding of this substance has given us doubly good results. In human medicine we use alkalies (Carlsbad water and salt), opium, phlegic acid, salicylate of soda. In order to sweeten the taste of food we may use saccharine.

DIABETES INSIPIDUS: SIMPLE DIABETES: PISSING.

Etiology. In human medicine we designate under the name diabetes insipidus an essential disease which is characterized by an excessive secretion of a very watery urine and which does not contain any sugar (whence the name diabetes insipidus). This disease appears to be dependent upon nervous disturbances, but its causes are very imperfectly known. It has been ascribed to injuries of the brain and several acute processes. It may be determined experimentally by a lesion of the rhomboidal sinus in the neighborhood of the pneumogastric centre of origin. If in some cases diabetes mellitus follows diabetes insipidus the degree of affinity of these two diseases remains to be elucidated.

The existence in our animals of a diabetes insipidus which is identical with that of man is not demonstrated. In veterinary medicine we have designated to the present time under the name of "pissing" the different forms of polyuria. But *pissing* is essen-

tially distinguished from diabetes insipidus by certain characteristics and especially by the extreme diversity of its causes. This is a manifestation which is common to a number of morbid conditions: it may be produced by the absorption of exudates, or it may occur either at the period of resolution of certain acute diseases, or during the course of diseases of the medulla oblongata, cerebellum, and kidneys; it is quite often occasioned by chronic interstitial nephritis (atrophied kidney) or renal hyperemia.¹ It appears also after the ingestion of large quantities of fluid, after the administration of diuretics, and in the sheep in poisoning by *Cynanchum vincetoxicum*. The principal cause of *pissing*, which is often recognized in an enzootic state in the equine species, is the ingestion of mouldy oats which have been rendered noxious by remaining a long time in the same place (ship's holds) and tainted without doubt by fermentations which are there produced by moulds. Damman has observed this polyuria after the ingestion of mouldy peas. Very likely he refers here to a poisoning which is complicated by hyperemia of the kidneys, or producing in these organs vasomotor troubles with increase of blood pressure. But toxic polyuria is also a different disease from diabetes insipidus.²

¹ Pulmonary tuberculosis of the horse is sometimes accompanied by an abundant polyuria which may reach as many as 18, 20, or 30 litres of urine, and which persists for some weeks (Nocard).—N. D. T.

² Moiroud, U. Leblanc, and Cagnat have mentioned interesting observations of enzootic polyuria. Moiroud reports that in 1830, in Paris and in certain regions of France, a large number of horses were affected by this disease. In several districts of Paris it attacked more than two-thirds of the equine population. It affected, in preference, heavy draught stallions, those which were employed for the transportation of building material, and especially plasterers' horses, in great numbers. Rare cases only were observed in livery stables and in pleasure animals. The disease was attributed to the use of tainted food and to the dampness of the atmosphere. In some stables, when it appeared, the horses were being fed on badly-harvested, muddy, and dusty hay, and with germinating oats which had a mouldy smell. As symptoms they recognized particularly: depression, exhaustion, diminution of appetite, heat and dryness of the mouth, frequent micturition, and insatiable thirst, acceleration of the circulation, and hardness of the pulse. Many patients urinated four, five, or six times per hour, and ejected about one litre of liquid at every micturition. In the beginning this was done in an easy and indolent way, later it became painful, a phenomenon which was attributed to a tumefaction of the urethral and vesical mucous membranes. The urine was limpid, of straw-yellow color, and of a density of 1007. The disease reached its height toward the tenth day; it remained stationary for a few days only; soon the symptoms became milder and a recovery occurred within three or four weeks. In some exceptional cases it has terminated in death. The only constant alteration which was found at the autopsy was an inflammation of the vesical mucous membrane. (See Recueil Vét., 1830.)

At St. Denis and in its neighborhood Cagnat has frequently recognized polyuria

However, in the horse we may meet with cases of chronic polyuria that last for years; they are incurable, their causes remain unknown, and in such no characteristic anatomical alteration exists. These cases only offer some analogy to diabetes insipidus of man. Stockfleth claims to have observed in the horse true diabetes, which was determined by cold. Perrin also recognized it in a horse which had received a kick on the anterior abdominal region, upon the surface of the liver.

Symptoms. From a symptomatic standpoint pissing of the horse corresponds exactly to diabetes insipidus of man. A few days after the ingestion of tainted oats the animals show disturbances of the appetite, which are sometimes accompanied by colics (Dinter). Soon micturition becomes more frequent and abundant. In twenty-four hours the patients eject 25 to 50 litres of urine; this is very clear, watery, and poor in solid substances; it contains neither albumin nor sugar; its reaction is acid when there are at the same time intestinal troubles; its specific gravity is diminished, it ranges between 1001 and 1015. The increase of urinary secretion occasions intense thirst; some horses have been seen to ingest 80 to 100 litres of water in twenty-four hours. The temperature remains ordinarily normal, but if serious gastric troubles occur we may recognize a more or less intense fever. The patients manifest all the symptoms of marked weakness.

In the majority of cases these symptoms disappear rapidly as soon as the unwholesome oats are withheld; in other instances, when toxic substances have acted for a long time upon the organism, a cure is difficult to obtain even when it is possible; occasionally emaciation becomes extreme, and the patients die in cachexia.

in heavy draught animals (plasterers' horses and those belonging to contractors of public highways). In most cases he has seen all the animals of the same stable become affected successively. The particular symptoms which he mentions are: dislike for oats, while the patients continued to take fodder, bran, and carrots, constipation, and an earthy shade of the conjunctiva. Concerning the etiology, he ascribes as a cause "contagion" or an intervention of infectious agents, and shows that the food and drink could not be held responsible. (See Archives Vétérinaires, 1884.)

Cagny has frequently found isolated cases of polyuria in horses during training. In 1883 he saw this disease exist in an enzootic state from the months of June to September. When the weather was cold the patients appeared to be livelier and more energetic, and the appetite returned; but in the majority of cases a final recovery only occurred with a lowering of temperature. "In some stables the appearance of *pissing* has followed the arrival of a horse which was already affected." (See Bull. Soc. cent. Vét., 1883.)—N. D. T.

Sometimes death occurs within a few months ; in other cases only after years.

Treatment. We must at once change the oats or render them harmless by means of cleaning and shaking, which ought to be repeated frequently. For a long period we administered internally certain astringents (acetate of lead, sulphate of iron, etc.), the efficacy of which is uncertain. In diabetes insipidus of man, opium (to calm thirst), valerian, and ergot of rye have rendered useful services ; we may try these in cases of obstinate chronic polyuria of the horse.

OBESITY.

NATURE. Obesity is an abnormal accumulation of fat in the organism, but especially in the subcutaneous connective tissue. It is observed in a physiological state during the process of fattening of certain improved breeds ; it is recognized as a pathological condition in draught animals and in the dog. It is in the latter that the veterinarian has usually to treat it. It does not assume near the same importance in veterinary as in human medicine.

Etiology. Over-feeding and want of exercise or insufficient work are the principal causes of obesity. A large number of house dogs "are, in fact, fattened by their owners."

Among breeding animals (bulls, boars), which are kept in the stable and abundantly fed, many become fat. The fat deposited is formed at the expense of the albumin, fatty bodies, and hydrocarbons contained in the food. Hydrocarbons favor its production in an indirect way ; by becoming decomposed in the organism they prevent tissue waste by saving the fat of nutrition. In the pig, ox, sheep, and perhaps also in the dog, obesity is sometimes developed as a consequence of an hereditary predisposition.

Anemia favors a deposition of fat in the organism ; this is a fact taught by experience, and is taken advantage of in an empiric way, in the beginning of the fattening process, by repeated bleeding.

Symptoms. Fat animals have rounded forms and an appearance which pleases the eye. The adipose pannicle is much developed ; in some animals the skin forms voluminous swellings which alternate with furrows ; others, especially the King Charles dogs, are often deformed by fat. In proportion to the increase of this substance vigor and vitality diminish ; the animals are lazy, weak, and rapidly fatigued ; disturbances of appetite and digestion appear ;

breeding animals become impotent. In excessive obesity we see dyspnoëic symptoms and an acceleration of the pulse, palpitations, and circulatory troubles (hypertrophy of the heart, infiltration and fatty degeneration of the myocardium).

Treatment. Obesity may be combated by different means:

1. By reducing the quantity of the ration or lessening the proportion of some of the alimentary matters of which it is composed.
2. By accelerating fatty decomposition.

1. *Lessening of the whole ration* is the simplest and most certain way; it always suffices in veterinary medicine. We have invariably succeeded in reducing the dog's condition through the diet; large-sized animals lose an average of one pound weekly.

2. *Suppression of certain adipogenous food*, such as fat and hydrocarbons, has been tried under various forms in man. It is a rational procedure, while it is somewhat artificial. The oldest method is that of Banting; this physician excluded almost entirely hydrocarbons as well as fat, and prescribed exclusively a meat diet (albuminoids). If used with moderation during a relatively short period, this treatment gives good results, but if continued too long the organism becomes unable to digest such considerable quantities of albumin; the animals soon feel an aversion to meat, gastric and intestinal catarrh are developed, weakness and exhaustion occur. In these conditions the exclusively animal food not being utilized, the organism exhausts its own albuminoids (the quantity of which is already insufficient in fat animals), because the fats and hydrocarbons always present in common food are absent.

In Epstein's method the hydrates of carbon are reduced to a minimum in the ration, which contains a very small proportion of fatty matter. Fat seems to diminish the sensations of hunger and thirst, and favors an assimilation of albumin.

3. *A destruction of fat* is especially produced by work and different physical exercises. By muscular activity the fat is consumed, acceleration of the circulation determines more rapid organic changes, and the energy of the myocardium is increased. The effects of this treatment have been known for a long time by horse-trainers. In man, Oertel considers it as the best method in order to combat circulatory troubles which occur during the development of obesity. He has also recommended a reduction in the quantity of drinks to the limit of sufficient allowance: a diminution of the quantity of water ingested eases the circulation, relieves the heart

(lessening of the blood-mass), and hastens organic metamorphoses; this treatment is therefore particularly adapted to cases of serious circulatory troubles; they may besides be associated to the muscular activity. The *ensemble* of these indications is commonly known as Oertel's method.

Formerly we made frequent use of bromides, alkalies, purgatives, and diaphoretics; but these agents, which are not used at the present time, sometimes exceed their aim, and may determine a gastro-intestinal catarrh.

SCROFULOSIS.

NATURE. In domestic animals scrofulosis does not constitute a definite nosological species; it represents, on the contrary, a group of morbid conditions. In ancient works authors describe it by assigning to it as principal symptoms: tumefaction, induration, suppuration, and caseation of the external and internal lymphatic ganglions, catarrhs of various mucous membranes, cutaneous eruptions, emaciation, serious troubles of nutrition, and, lastly, cachexia. Most cases of scrofulosis mentioned in veterinary medicine seem to be connected with tuberculosis, as has already been pointed out by Spinola; others belong to rhachitis, to infectious pneumonia (especially in the pig), to parasitic diseases of the lungs (*Strongylus paradoxus*), to pyemic polyarthritis (in the colt), to metastatic strangles (formerly strangles were called by the name of *Scrofula equorum*); others, again, in aged animals, appear to be dependent on leukemia, pernicious anemia, chronic intestinal catarrh, or cachectic diseases of different nature. In the foal scrofulosis has been designated under the name of *consumption* (*Darrsucht*). But the latter disease is generally considered as an intestinal tuberculosis.

We have mentioned *scrofulosis* and *consumption*, so as to conform to custom, and in order to show that it would be of advantage to abandon these old denominations, which are still used by some practitioners.

SARCOMATOSIS AND CARCINOMATOSIS.

Sarcomatous and carcinomatous neoformations are particularly interesting from a clinical standpoint when they are developed in

the liver, stomach, intestine, kidney, uterus, lungs, brain, etc., or when they are generalized. In the horse and dog sarcomata and carcinomata frequently develop with the clinical tableau of peritonitis or chronic pleurisy, which are accompanied by an abundant serous or hemorrhagic exudate.¹ Vast cancerous neoplasms may remain localized for a long time upon the pleura and the peritoneum. In the ox sarcoma of the abomasum is marked by permanent disorder of digestion and nutrition. In the female dog carcinoma affecting the mammæ is frequently accompanied by secondary tumors in the abdominal and thoracic organs, uterus (bloody, fetid vaginal discharge), liver, lungs (symptoms of pulmonary phthisis), etc. Ulcerating sarcomata and carcinomata are

¹ The mode of development of malignant tumors, their progressive local extension, the invasion of the lymphatic ganglions, and the metastatic phenomena which they produce have for a long time made us suspect their infectious parasitic nature. Grafting and inoculations have been practised with the object of reproducing these tumors and to demonstrate their transmissibility. Almost all these attempts have been unsuccessful. Only of late years have a few positive results been obtained. Hanau (of Zurich) has inoculated with success the carcinoma of a rat in two other animals of the same species by inserting a portion of the tumor into the vaginal sheath. Both of the inoculated subjects were dead within seven weeks. At the autopsy there were found upon the epiploon tumors, the cancerous nature of which was demonstrated by a histological examination (Medicin. Congress, Wiesbaden, 1889). In mice, Morau has succeeded in grafting, in the axilla and groin, fragments of an epithelioma which came from an animal of the same species. The tumor was reproduced with its histological characters. Graftings in series which have been performed on eighteen subjects have invariably given positive results, and in some cases authors have obtained visceral generalizations (Bull. de la Soc. Biologie, 1891).

In the human species Hahn (of Berlin) has mentioned the fact of a cancerous transplantation to a healthy region in a patient affected by incurable cancer (Medicin. Congress, Wiesbaden, 1889). Two other observations of the same kind were reported a few weeks ago to the Academy of Medicine. One case was a fasciculated sarcoma of the breast, and the other an epithelioma of this organ. Grafting of a fragment of these tumors, which was practised with the *most minute antiseptic precautions*, in the opposite mammary gland, which was healthy, was followed by development of neoplasms of the same nature. Such are the facts which have been obtained concerning this important question. They establish that a fragment of sarcomatous or cancerous tumor placed in contact with the normal tissues of an affected subject, or of an animal which is apt to contract it, becomes fixed in these tissues and is developed by invading them. They enable us to understand how the cells of a peritoneal or pleural neoplasm, if they are slightly adherent to its surface and in connection with other normal points of this serous membrane may become grafted into it and determine numerous secondary neoplasms. Finally, they give an explanation of the generalization of some tumors through vascular ducts; the lymphatic or blood currents carry the cells produced by these tumors, which become grafted in the neighboring ganglions or in the viscera.

If specific micro-organisms exist in the malignant neoplasms, they have up to this time escaped the investigations of bacteriologists. The discovery of the bacillus of cancer, asserted by Scheurlen (of Berlin) in 1887, has not been confirmed.—N. D. T.

often complicated by a serious septic fever. When these neoplasms exist for some time they affect nutrition; the animals lose flesh and become cachectic (cancerous cachexia). As soon as they reach the viscera they become fatal. No treatment can arrest their course. All that can be done is to sustain the organism by nutritious alimentation and increase its resistance to the invasion of morbid elements by the daily administration of small doses of arsenious acid or of Fowler's solution.

SECTION V.

EPIZOOTIC AND INFECTIOUS DISEASES.

GENERALITIES. Epidemic and infectious diseases are caused by pathologic microbes. In some the specific agent is well known (tuberculosis, anthrax, etc.); in others it is yet to be discovered (hydrophobia, canine distemper). The contagiousness of these diseases, their incubation, and their transmission to healthy animals by the action of infinitesimal quantities of virulent matter, can only be explained by admitting the intervention of a living contagion. The number of known pathologic microbes increases every day, and no doubt in the near future the obscurity which still covers the pathology of certain infectious diseases will be dissipated.

These diseases have been divided into *contagious*, *miasmatic*, and *miasmatico-contagious*. An infectious disease is said to be *contagious* when the agent which produces it may be carried from a sick animal to a healthy subject, either directly or by some intermediary; it is called *miasmatic* when the virulent agent originates according to circumstances (soil, water from certain localities); it is finally qualified *miasmatico-contagious* when, being of miasmatic origin, the disease is transmitted from affected animals to healthy ones. Pettenkofer distinguishes *endogenous* and *exogenous* infectious matter; while the latter have an extra-organic origin, the former are said to be generated in the organism itself. We shall not insist upon these divisions; they are of only secondary interest.

The biological study of the pathologic schizomycetes is extremely important from the standpoint of treatment of infectious and epizootic diseases. We must here limit ourselves to making a summary study of the biology of microbes, and refer the reader to special publications.¹

¹ Lehrbücher der Bacteriologie, by Zopf, Flügge, and Marpmann; Jahresber. über die Fortschritte in der Bacteriologie, by Baumgarten; Die Pflanzlichen Parasiten. by Zürn: Die Veröffentlichungen des deutschen Gesundheitsamtes. Among French works see, especially, Cornil and Babes: Les Bactéries, 2d édit., Paris, 1890; Arloing: Les Virus, Paris, 1891; Gallier: Traité des maladies contagieuses des animaux do-

The microbes (schizomycetes or bacteria) are nearly all simple unicellular vegetations deprived of chlorophyll. They exhibit the most varied forms: Micrococci or spherobacteria, small sticklets or *Microbacteria*, lengthened sticks or *Desmobacteria* (bacilli), spiral elements or *Spirobacteria*, with the three lower species—*Vibrio*, *Spirillum*, *Spirochaete*.

The first-known pathologic microbe, the bacteridium anthrax bacillus (*Bacillus anthracis*), was discovered almost at the same time by Pollender (1855) and by Branell (1857). Davaine declared it to be the specific agent of anthrax.¹

The reproduction of microbes is performed at times by segmentation, at other times by sporulation (within the mother cells female cells are formed, which are very refringent—spores—which become free after the death of the first). In order to feed and become developed the microbes make use of organic substances, mineral salts, and water, and they need a certain temperature; oxygen, which is indispensable for some, is toxic for the others; hence the division into *aërobic* and *anaërobic* microbes (Pasteur).

For bacteridia the most favorable temperature is found between $+30^{\circ}$ and $+40^{\circ}$ C.; its development is arrested above 42° and below 15° C. Boiling water (100° C.) and hot air at 140° C., acting for a sufficient length of time, kill the bacilli and spores.²

mestiques, Paris, 1891; Charrin: art. Pathologie général infectieuse du Traité de médecine, Paris, 1891.

¹ In 1850 Davaine and Rayer remarked in blood infected with anthrax the presence of small filiform bodies nearly double the length of the diameter of a blood corpuscle, and which did not show any spontaneous movements.

² Most microbes possess such a resistance to the action of cold that the lowest temperatures which exist upon the surface of the globe are powerless to destroy their vitality. Arloing, Pictel, and Yung have been unable to destroy the fertility or the virulence of the *Bacillus anthracis* and the *Bacterium Chauvœi* by treating them as follows: Exposure for twenty-four hours to a temperature of -70° C., which is produced with liquid sulphurous acid; for eighty-four hours to -70° to -76° C., which is produced with solid carbonic acid at ordinary pressure; for twenty hours to -120° to -130° , which was produced with solid carbonic acid liberated in a vacuum. But we may destroy microbes by high temperature. "In the state of organic vegetation microbes do not resist a temperature above $+100^{\circ}$ C. With rare exceptions the micrococci are killed between $+50^{\circ}$ and $+60^{\circ}$, and the bacilli between $+70^{\circ}$ and $+125^{\circ}$. If the microbes contain spores or arthrospores, they are killed only between $+110^{\circ}$ and $+125^{\circ}$. Tyndall has remarked that *three hours* of continuous *boiling* did not sterilize an infusion of hay in which several germs were sporulated or in a condition of spores, which *three minutes* of repeated *boiling* for three consecutive days, once a day, was sufficient to insure the preservation. This strange result is accounted for by the fact that during the interval between the boilings the spores germinated and passed into a state of mycelium, which was less resistant to heat than the forms of rest." (See Arloing's Les Virus, Paris, 1891.)—N. D. T.

We find bacteria wherever organic substances exist, but especially in the animal and vegetable tissues, in putrescent or fermenting liquids (the experiments of Pasteur on putrefaction and fermentation have been the starting-point of modern bacteriology). We meet with some in running water, stagnant water, in the superficial layers of the soil (as deep as 1 metre), and in the air. Bacteria penetrate into the organism with food, drink, air, and by wounds of the cutaneous or mucous surfaces. They exercise their poisonous action by appropriating from organic matters the oxygen which is necessary to their nutrition, and in generating toxic products (putrid principles; cadaveric ptomaines or alkaloids).

There are numerous chemical agents (antiseptics or disinfectants) which are capable of hindering the development of microbes and destroying them. The principal are: sublimate, which, in a solution of 1 : 300,000, arrests the development of anthrax spores, and in a solution of 1 : 20,000 kills them in the space of ten minutes; the essences, especially essence of peppermint, which, in a dilution of 1 : 300,000, prevents the development of the spores, and essence of turpentine, which possesses the same property in a solution of 1 : 75,000.

Among other microbicides we must particularly mention iodine (1 : 5000), bromine (1 : 3000), chloride (1 : 25,000). According to Eisenberg, cresol in a solution of 1 : 15,000 arrests the development of anthrax spores;¹ these resist phenicated water in an 8 per cent. solution for seven days, while 3 per cent. cresol water kills them in forty-eight hours. Antiseptics play an important rôle in the prophylaxis of infectious diseases; but up to the present time they cannot be used for the treatment of these affections; some even weaken the constitution and have a harmful influence.

Vaccinations seem to be destined to occupy a very important place in the prophylaxis of infectious diseases. They are based upon the principle that a first infection by virulent agents generally confers a relative or absolute immunity.

This preventive method, which is of recent origin, will remain under investigation for a long time yet, but in some diseases it has already given incontestable results. This is practised by means of a great variety of manipulations: prolonged artificial culture

¹ Lignières has ascertained that dried-up anthrax spores are not destroyed by immersion in a 5 per cent. solution of cresol which is prolonged for twenty days.—

(anthrax, chicken cholera), high temperature (symptomatic anthrax), transfer of the virus to another species of animals (vaccination, hydrophobia, swine plague of the pig); by a change of the nutritive centre or a prolonged culture in the same liquid, dilution, addition of chemical agents, special process of inoculation, we obtain a weakening of the activity of the infectious principle, which preserves, however, its generating biological properties of immunity. Lately investigators have attempted to prevent certain epidemic diseases by inoculation of the virus of other specific diseases.

Emmerich has succeeded in conferring upon some animals immunity against bacterial diseases and other pathologic microbes by inoculating them with the micrococcus of erysipelas. But before assuming the advantages which may be drawn from this discovery we must wait for the results which a thorough study of the question will furnish.

A knowledge of the etiology of infectious diseases has entirely transformed the opinions formerly entertained on this subject, and every day new discoveries come to enrich the vast field of the inner pathology. At the present time we cannot give a complete description of these diseases; we have done our best to explain here the actual state of science.

SEPTICEMIA AND PYEMIA.

GENERALITIES UPON THE TERMS SEPTICEMIA AND PYEMIA.

1. Modern authors use the word *septicemia* in order to designate certain infectious diseases in which the blood is overrun by micro-organisms, and which are characterized anatomically by tumefaction of the spleen, as well as by various degenerative lesions of the parenchymatous organs (liver, kidney, heart). Nevertheless, we must remark that these anatomical alterations are also observed in anthrax, swine plague, chicken cholera, and the epidemic disease of wild animals (*Wildseuche*), etc. These diseases must be considered as *specific septicemia*, having a special denomination and being produced by particular micro-organisms. Besides these specific processes there exist simple septicemias, the pathologic agents of which are little known. Among these latter we count surgical diseases, which are designated by the denominations of *septicemia*, "*septhemia*," *septico-pyemia*, *putrid fever*, etc., which occur as complications of

wounds or ulcerations. When these morbid processes are connected with internal diseases (gangrenous pneumonia), they are known as *spontaneous*, *primary*, and *cryptogenetic*. We are aware of but a small number of pathological bacteria which occasion a simple form of septicemia in domestic animals; in some instances they are *cocci*, in others *bacilli*. Davaine has found in the rabbit a septicemia which was produced by micrococci; Koch has described a bacillary septicemia of the mouse; Gaffky has discovered another in the rabbit. Koch's malignant œdema, for which we reserve a special description, is an infectious complication of wounds, which is produced by a fine, mobile, and characteristic bacillus (Pasteur's septic vibron). The septicemic process is complex and multiform; it varies with the animal species, and in the same species we may meet with several simple septicemias.

2. *Pyemia* is an infectious disease which is occasioned by pyogenic micro-organisms. Like septicemia, pyemias are *specific* or *simple*. Strangles of the horse, which is determined by a special pyogenic streptococcus (Schütz), must be considered as a specific pyemia. Surgical pyemia is a simple pyemia; it is produced by common pyogenic micrococci; in man, in the pus of metastatic abscesses, Rosenbach has especially found the *Staphylococcus albus* and *aureus*. It has generally as a starting-point venous thrombi which are undergoing putrid decomposition within traumatisms on the mucous membrane (in the "puerperal" uterus), in the lungs, the feet, and in bony wounds, or purulent centres developed within the organs (lymphatic ganglions which have formed abscesses during the course of strangles, pharyngitis, etc.). Omphalogenic pyemia of newborn animals and metastatic accidents which are connected with parturition have been described previously.

In order to produce pyemia it is sufficient that pyogenic micro-organisms penetrate into the blood; the intervention of purulent corpuscles is not necessary.

The preceding considerations show that the specific microbes of septicemia and pyemia in domestic animals are but imperfectly known. Prof. Schütz, who has made this question a subject of special bacteriological research, has sent us the following communication: In the horse, in cases of common abscesses, we find especially the *Staphylococcus albus* and *aureus*, more rarely other pyogenic micro-organisms. The same micro-organisms exist in pyemic abscesses of the internal organs. In the purulent collection of

strangles we find the characteristic streptococci of strangles. There is no essential difference between pyemia and septicemia; in the latter, as in the former, the blood centre is infected by the pyogenic microbes. The clinical peculiarities of these processes are due essentially to the differences in quantity and the virulence of the pathologic agents which are introduced into the blood.

Pathological anatomy. 1. In *septicemia* the most important anatomical alterations are those of the blood; this liquid is uncoagulable and rich in bacteria; its surface is varnished-like. The myocardium, kidneys, liver, etc., have undergone fatty degeneration; the spleen is tumefied; we find hemorrhages in most of the organs and sometimes a septic endocarditis. In some cases the microbes are accumulated in colonies; this is a frequent peculiarity in the kidney (bacterial nephritis). We observe numerous micro-organisms which are fixed upon the walls of the capillaries; they often completely obstruct part of these canals; the leucocytes sometimes contain bacteria. The cadavers become decomposed very rapidly. When the disease has followed a subacute course the alterations are frequently insignificant and may pass unnoticed.

2. The alterations of *pyemia* have for a long time attracted the attention of veterinary writers. Renault and H. Boulay, after having studied the effects of intravenous injections of pus (1830–1840), have succeeded in establishing a theory, claiming that glanders may be produced by purulent absorption. In pyemia the blood contains pyogenic micro-organisms; abscesses are found in most of the organs (“multiplex” or metastatic pyemia), in the lungs (*pus buttons* (green abscess) of old authors), liver, kidneys; we find purulent phlegmasias in the serous membranes (peritoneum, pleura, meninges, articular, synovial), in the eye (purulent choroiditis and panophthalmia); sometimes there exists an ulcerative endocarditis and numerous small hemorrhages upon the serous membranes, the skin, eye, and muscles, etc. Quite frequently we recognize also alterations of septicemia (septico-pyemia).

Symptoms. 1. The clinical aspect of *septicemia* is outlined by very marked pyretic phenomena, by a temperature which reaches and even exceeds 42° C., by chills, a small, accelerated pulse which later becomes imperceptible, and by atony of the heart. We observe serious general troubles: extreme weakness, drowsiness and stupefaction, icteric coloration and ecchymoses of the mucous membranes; urinary troubles, albuminuria, sometimes hemoglobinuria, complete

loss of appetite, and later profuse diarrhea. The duration of the disease is very variable. Death may take place within a few hours, but ordinarily the affection continues several days; it may even be prolonged for weeks. Recoveries are rare.

2. The symptoms of *pyemia* are occasioned by the metastases and by the phlegmasias which accompany them. Fever is high, intermittent, and accompanied by chills. According to the cases, we observe the symptoms of metastatic pneumonia, with its ordinary termination of pulmonary gangrene, or those of hepatic, renal, and cerebral abscesses; there is often pleurisy, meningitis, polyarthritis, etc.; we may see numerous subcutaneous abscesses. During the course of pyemia a "hemorrhagic diathesis" generally occurs, which is marked by blood suffusions in the viscera and in the mucous membranes. According to the locality and extent of the metastasis, the duration of the disease varies from a few days to several weeks. While it is more common than is septicemia, recoveries are nevertheless rare; convalescence is always slow. Pyemia produces lesions which vary extremely and are marked by a very complex symptomatology. We cannot possibly give a general description of it.

Diagnosis. A diagnosis of spontaneous non-traumatic septicemia is far from being always easy. Intense fever and serious general troubles are the most important points of evidence on which we may depend in cases where no primary local disease exists. It may be confounded with other febrile processes and with anthrax (see Differential Diagnosis of Anthrax).

A diagnosis of pyemia is based upon the existence of an intense fever, metastatic or embolic inflammations, and suppuration in the different organs; as a rule we find also a primary purulent centre. It may be confounded with glanders (see Differential Diagnosis of Glanders).

Treatment. The treatment of septicemia consists of the administration of antiseptics and of antizymotics; we recommend especially camphor, quinine, essence of turpentine, and calomel in small and repeated doses. We administer camphor internally or in subcutaneous injections, in the form of oil or of camphorated alcohol; if associated with alcohol or ether, it may act favorably in cases where weakness is very pronounced. We may also try the different modern antipyretics. In traumatic septicemia local antisepsis is the main indication.

The treatment of pyemia differs little from that of septicemia. We generally have recourse to the same therapeutic agents. Internal medication offers little chance of success. The intervention ought to be mainly surgical.

MALIGNANT ŒDEMA. (KOCH.)

Etiology. Among the septicemias which have been mentioned Koch's malignant œdema (Pasteur's gangrenous septicemia) offers a particular clinical importance. It is determined by small sporogenous bacilli (œdema bacilli, *Vibrio septicus* of Pasteur), which exist in large numbers in nature, and especially in the superficial layers of the soil. If garden-earth is placed under the skin of a rabbit, the animal generally dies from malignant œdema within twenty-four to thirty-six hours. Infection is easily produced when the septic vibrio is injected into the subcutaneous connective tissue; if introduced into the blood, this anaërobic microbe is innocuous—it is killed by the oxygen (Chauveau). Inoculation into the derma is also without effect. Granulating wounds constitute an insuperable barrier. According to Chauveau, a first infection confers immunity.

Morphologically the *Vibrio septicus* resembles somewhat the bacteridium, but it is thinner, finer, and sometimes animated by rapid movements; its extremities are rounded. After the death of the septicemic animal it acquires a great length and forms stiff filaments, which are slightly incurved or handle-shaped; it is often formed of articulated segments, and then presents the appearance of a little chain. The spores are developed later at the expense of these filaments. Vibrios are found in great quantities in the blood of asphyxiated animals which have been exposed for twenty-four hours to a temperature of 38° C.; they are especially numerous in the blood of the portal system; the temperature of the central parts of large-sized cadavers falls but little after death from dyspnoea, colics, etc.; they are found at first in the blood of the liver, and in the whole organism after a certain time (twelve to twenty-four hours). Lustig, who was the first to remark this fact,¹ admits the possibility of a spontaneous infection of the horse

¹ The invasion of cadavers by the *Vibrio septicus* contained in the intestine has been distinctly mentioned by Pasteur and Joubert in their work *Anthrax and Septicemia*, which was communicated to the Academy of Medicine in 1877.—N. D. T.

by *Vibrio septicus* coming from the intestine; he has related a few observations.

The researches of Kitt have established the fact that malignant œdema is transmitted by inoculation to the animals of our various domestic species (guinea-pig, goat, calf, sheep, dog, horse, chicken, pigeon).¹

¹ Bottini was the first who recognized the inoculability of surgical septicemia. Billroth has demonstrated experimentally that the septic elements easily invade the superficial layers of recent wounds, but that they are powerless to force a way through the tissue of granulations.

This septicemia is produced by the *Vibrio septicus* of Pasteur. This micro-organism is perfectly developed in the air, in carbolic acid, and in inert gases; it is killed by an excess of oxygen in the culture liquid. But the septic elements are only destroyed in liquids which are spread in a thin layer; when the layer is of a certain thickness the vibrios of the lower layer are multiplied at first by segmentation, then they end by becoming resolved into germ corpuscles or spores, which resist oxygen and the majority of antiseptics. They are found in the earth, water, air, on the surface of solid bodies, etc. The *Vibrio septicus*, being anaërobic, acts like a ferment. As long as the power of multiplying by segmentation lasts it is accompanied by the formation of carbonic acid, hydrogen, and putrid gases; these gases, which explain the local emphysema and the rapid cadaveric swelling, are no longer produced when the transformation of the vibrios into germ corpuscles is accomplished. In contact with oxygen, the latter remain sterile; protected from its action, their activity becomes apparent: they are transformed into vibrios, and their pullulation begins. When septic elements are deposited upon regular or anfractuous recent wounds, their noxious effects may be prevented by irrigation with aerated water or allowing plenty of atmospheric air to reach their surface; under these conditions the vibrios are killed and the activity of the spores is restrained. But if a blood-clot or a shred of mortified tissue protect these agents from the influence of oxygen, a condition is produced favorable for a beginning pullulation and septicemia (see Pasteur, Joubert, and Chamberland: "The Theory of Germs and its Applications to Medicine and Surgery," *Compt. rend. de l'Académie des sciences*, 1878).

Chauveau and Arloing have established that in man and animals susceptible of contracting gangrenous septicemia this disease is determined by Pasteur's *Vibrio septicus*. This micro-organism presents peculiar characters in the connective tissue and serous membranes. In œdema of septic centres it is found either in the form of a bacillus provided with a spore at one of its extremities or as a bacillus of homogeneous protoplasm a little longer than the other. In the serous membranes it reaches a considerable length: later it becomes segmented into non-sporulated sections. It invades the circulatory blood system only toward the end of the disease or after death; it is found in the blood in the form of a short bacillus or as a common micrococcus. Connective tissue which is protected from air constitutes the most favorable medium for the inoculation of gangrenous septicemia. The vascular blood system offers a remarkable resistance. If, after having forced a few drops of virulent serum into the jugular vein of a ram the circulation in one of its testicles be interrupted by the operation of bistournage, this organ becomes the starting-point of a fatal gangrenous process. This is also the case if the virus introduced into the blood penetrates into the network of the connective tissue by means of a rupture of the blood-vessels; without external wound it may determine all the phenomena of gangrenous septicemia. Septicemic discharges gradually lose their noxious properties in direct

Symptoms and anatomical alterations. In malignant experimental œdema an œdematous, doughy, sensitive, and crepitant tumefaction appears at the point of inoculation, and extends by degrees in all directions. The reaction is intense. The subcutaneous connective tissue and the adipose tissue, as well as the muscles in the neighborhood, become the seat of a gelatiniform infiltration; a large quantity of very fetid gaseous bubbles is developed therein; the reddish-yellow liquid of the œdema contains numerous characteristic bacilli, which are wanting in the blood during life and which are found in it in only very small numbers after death. [In addition to these local symptoms we will notice as a characteristic phenomenon the cold, moist, clammy condition of the central portion of the mortified tissue, while the peripheric portions are hot, tense, and extremely sensitive; the *Vibrio septicus* has apparently abandoned the first region, as only putrefactive germs can be found therein, and has invaded the surrounding tissues, wherein it seems to live at the expense of the vitality of the cellular elements with which it comes in contact; at this stage an invasion of new tissue is again observed, and so on in regular progression until general infection results.—W. L. Z.] There exist at the same time pulmonary œdema and a phlegmasia of the mucous membrane of the intestinal grêle. The serous liquid discharged into the pulmonary alveoli contains numerous bacilli. The spleen, liver, and kidneys are not attacked; splenic tumefaction is wanting. The disease generally terminates in death within a very short time (twenty-four to forty-eight hours). The inoculation of small quantities of septic vibrios may end in a cure after the formation of a local abscess.

proportion to the progress of putrefaction; within two months their virulence is generally exhausted, but if dried between $+15^{\circ}$ and $+38^{\circ}$ C., and before putrefaction has set in, they preserve this virulence almost indefinitely. They are little affected by antiseptic agents; when in a fresh condition their virulence is destroyed by a temperature of 100° prolonged for fifteen minutes, and in a dry state by a temperature of 120° lasting ten minutes (see Chauveau and Arloing: "De la septicémie gangreneuse," Bulletin de l'Acad. de méd. et de la Soc. cent. Vét., 1884).

Chamberland and Roux have succeeded in conferring immunity to animals by means of inoculation in the peritoneum of soluble *vaccinal* substances. They have used prepared *Vibrio septicus* cultures, in which the microbes had been killed in a temperature of 105° to 110° C., which was prolonged for ten minutes, or the liquid which was obtained by filtration of these cultures. The degree of immunity which was thus conferred was in direct proportion to the quantity of injected preserving matter (see Recueil Vét., 1888).

By combining the action of heat and that of antiseptics Cornevin has obtained vaccine which enables us to confer on animals, and particularly the dog, a safe immunity from septicemia. (Comptes rendus de l'Acad. des sciences, 1887.)—N. D. T.

Differential diagnosis. Kitt has pointed out the great symptomatic analogy which exists to malignant œdema and bacterian anthrax. The distinction between these diseases is based upon the enzootic character of symptomatic anthrax (which is a disease ending almost always in death) and upon the result of a bacteriological examination. [Another characteristic clinical symptom which enables us to recognize the malignant œdema of Koch is its appearance after a wound and the existence of well-defined local symptoms which precede the fever.—W. L. Z.] By reason of the dissemination of the *Vibrio septicus* malignant œdema may develop anywhere. We differentiate it from bacterian anthrax by the absence of micro-organisms in the blood during life, by the mobility of the septic vibrio, the rounded form of its extremities and the peculiarities displayed by its culture (see Differential Diagnosis of Anthrax).

Treatment. It is especially surgical. Its principal indications are extensive incisions in the emphysematous tumefactions, drainage, and disinfection of the subcutaneous connective tissue.

Kitt has shown that the bacilli of malignant œdema are probably the causative agents of a whole series of diseases in animals: septicæmia, several diseases of the ox similar to swine plague, certain pulmonary œdemas the pathology of which is obscure. Many of the cases of *septic parturition fever* and *surgical phlegmonous tumefactions*, “*progressive inflammation of the connective tissue*” of Haubner and “*the penetrating disease of the horse*” (*Einschuss*), etc., are but malignant œdema. The same is the case with “*sheep fire*,” which, according to Haubner,¹ has mostly as an exciting cause bites on the legs, but which appears sometimes upon the surface of bleeding wounds. It is especially observed in autumn, when the animals are subjected to a mixed regimen of pasture and dry food. The symptoms appear suddenly; they consist of an œdematous and painful tumefaction of the skin and the subcutaneous connective tissue, which is especially marked upon the inner fascia of the posterior legs, more rarely upon the anterior members, the neck and shoulders, or in the submaxillary space; the patients are lame, the febrile reaction is intense. Tumefaction of the legs extends very rapidly to the abdomen and chest, the skin becomes successively bluish-red, violet, or lead-colored; it crepitates under the hand; later, it is found completely mortified, cold, and insensitive; stupe-

¹ Haubner: Landwirthschaftl. Thierheilkunde, und Magazin, 1849.

faction and drowsiness increase; the animals are stretched upon the ground in an entirely inert condition, they die in from eighteen to twenty-four hours, more rarely within two or three days. When the disease is localized on the neck and shoulders we observe phenomena which are similar to angina; in some cases we notice diarrhea and bloody micturition. At the autopsy we find a hemorrhagic infiltration of the subcutaneous connective tissue, which is distended by gases; the cadaver decomposes very rapidly; there are hemorrhages in different organs, the blood is brownish and uncoagulable; the spleen is not tumefied.

We have observed the same disease in the dog after bites, and in the horse after castration. In the former it is not rare to recognize rapidly-growing œdematous tumefactions of the subcutaneous connective tissue, which are accompanied by septic emphysema and extended necrosis of the skin, and which always terminate in death. This disease seems to have been very frequent formerly as a complication of surgical operations which were practised with unclean instruments. It was confounded with anthrax.

PETECHIAL FEVER OF THE HORSE.

Typhus Fever of the Horse: Petechial Disease: Anasarca:
Morbus Maculosus: Purpura Hæmorrhagica.

ANCIENT THEORIES CONCERNING PETECHIAL FEVER. Ancient authors designated, under the name of *typhus*, *putrid fever*, all infectious diseases accompanied by decomposition of the blood and serious cerebral phenomena: anthrax, septicemia, influenza, contagious pneumonia, acute glanders, cerebral meningitis, etc. In a great many diseases they describe an inconstant "typhus" period, which is characterized by serious disturbances of the central nervous system. Petechial fever was considered as a variety of typhus, which was distinguished from others by being defined under the name of *typhus of the horse* or *petechial typhus*; these denominations have been preserved up to the present time. The expression *putrid fever*, which was used in 1836 by Hertwig, has been accepted by Spinola and a few others. Hering has designated the disease according to one of its principal symptoms—*petechiæ of the mucous membranes*; he pointed out at the same time that these lesions are observed not alone in petechial fever—for instance, in strangles.

The expression *typhus* has given rise to an acceptance by some authors of the identity of this disease of the horse and abdominal or petechial typhus of man. In England it has been likened to scarlatina and to the petechial disease or *morbus maculosus* of man (*purpura hæmorrhagica*). The appellation petechial disease was also used in Germany, where it was first employed by Eberhardt. More recently Dieckerhoff has again advocated its use. In France and Italy the disease is described at the present time as an acute dropsy of the derma and subcutaneous tissue [*anasarca* (Trasbot)]; it is attributed not to a decomposition of the blood, but to a temporary paralysis of the capillaries, followed by an exudation of serum and blood. Lafosse related it to septicemia, and Bouley to anthrax.

In Germany, Röhl was the first to consider petechial fever as a form of anthrax. Hering and Haubner had, however, shown that it is neither contagious nor transmissible by inoculation to the horse and to the subjects of other species, and that the bacteridium is never found in the blood. Arloing has not succeeded in transmitting it by the transfusion of the blood of diseased animals. Moreover, certain symptoms are unexplainable if we classify anthrax as a form of petechial fever.

Dieckerhoff has declared himself an advocate of the identity of this disease with *morbus maculosus hæmorrhagicus Werlhoffii* of man. He has proposed to give it the old name petechial disease, or *morbus maculosus*. We will preserve the term *petechial fever*, but without intending by this to establish any closer relation between it and the petechial disease of man, of which the nature and causes are little known, the essential nature yet to be established, and its relations with other hemorrhagic diseases not well defined. We agree with Dieckerhoff in rejecting the word *typhus*, which gave rise to the belief in the similarity of this disease to the typhus of man, which has absolutely nothing in common with petechial fever.

In man we recognize three varieties of typhus: 1. Abdominal or ileo-typhus. 2. *Exanthematic or petechial typhus*. 3. *Recurrent typhus*, also called *recurrent fever*. *Abdominal typhus* is produced by a specific bacillus (Eberth's bacillus). It is a miasmatico-contagious disease, which consists essentially of a necrotic infiltration of the lymphatic follicles of the intestinal grêle, which is particularly marked in the ileum, in the neighborhood of Baubin's valve.

The spleen is greatly tumefied; most of the mucous membranes show catarrhal symptoms. The disease is accompanied by chills; the mental condition is much affected; there is obstinate diarrhea, bronchitis, and serious nervous manifestations (delirium, convulsions); a few red isolated spots (three to six) appear upon the chest and the belly. We may observe the greatest variety of complications: enterorrhagia, intestinal perforation, angina, ulcers of the larynx, parotiditis, serious pneumonia, nephritis, cerebral accidents, emboli, paralysis, etc. At the present time the mortality reaches nearly 7 per cent. Several experimenters have made animals ingest the excrements of typhus men without obtaining any positive results. The experiments of Baumgarten have demonstrated that the typhus bacilli of man are harmless to rabbits, guinea-pigs, and mice.¹ None of the numerous observations of transmission of abdominal typhus to animals are convincing. Authors have no doubt confounded with anthrax (case reported by Ammon), mycotic gastro-enteritis, several septic processes, perhaps also a few intoxications. Semmer is the only veterinary author who admits the existence of the typhus of man in animals (horse, dog). (See Influenza.)

Petechial typhus of man, which is a very contagious disease, exists in an endemic state in certain countries (*typhus of inanition, army typhus*). At the autopsy we do not find any characteristic alterations; intestinal lesions are wanting. The course of the disease is typic; there is produced upon the surface of the skin a roseolar exanthema, which is followed by petechiæ or true hemorrhages. Improvement and recovery often take place with extraordinary rapidity. The mortality is about 10 per cent.

Recurrent typhus is determined by a specific spirillus (Obermeyer's spirochete) which is found in the blood. It also evolves without alterations. Its principal symptom is an intense intermittent fever. Its course is very mild (mortality, 2 per cent.). These last two varieties of typhus are never observed in animals.

Etiology and pathology. Petechial fever is an acute infectious disease, the pathology of which is as yet unknown. It is characterized by numerous petechiæ of the skin and subcutaneous and submucous connective tissues; also by petechiæ of the mucous membranes and internal organs; to these phenomena are soon added

¹ Chantemesse and Vidal have obtained different results. By inoculating rabbits, guinea-pigs, and mice with cultures of Eberth's bacillus, they have often produced, especially in the mouse, a fatal septicemia.—N. D. T.

tumefactions of the skin and mucons membranes and infiltration of the subcutaneous connective tissue. Sometimes it is primary ; in other instances it follows other infectious diseases : strangles, pharyngitis, contagious pneumonia, influenza, etc. Chronic pulmonary catarrh, chronic diseases of the maxillary sinus, intestinal catarrhs with purulent destruction of the lymphatic follicles ; abscesses of the lungs, kidneys, and spleen ; infected traumatism (castration wounds and others), have been pointed out as causes of petechial fever. In one case Siedamgrotzky has seen it develop after an insignificant wound of the skin (crack). Horses employed by excavators seem to be particularly subject to it. Nothing is really known of the nature of the morbid principle. Is it a chemical poison or a figurative element ? Dieckerhoff believes in an antipoisoning originating from primary morbid centres : suppurative adenitis, abscesses and purulent catarrhs. The pathologic agent is a chemical poison developed in these local lesions by the microorganisms ; in altering the vascular walls this poison determines hemorrhages. This chemical theory explains the development of the disease, which is frequently apyretic, and the simultaneous appearance of tumefactions in various regions, experimental results demonstrating that the disease is neither contagious nor inoculable, and also the failure of bacteriological researches. But the hypothesis that the toxic substance comes from primary morbid centres is disproved by the absence of these lesions in a certain number of cases where the disease originates in perfectly healthy animals. We must add that in the autopsy it is often very difficult, if not impossible, to recognize primary centres and to distinguish secondary centres, especially when the disease has been of long duration. Finally, the rarity of petechial fever in strangles and the chronic purulent catarrhs, when abscesses are so frequent, does not agree very well with that doctrine. The symptomatic analogy of the disease to malignant œdema, the enzootic character which it takes at certain times (Zschokke has seen true enzootics in recruiting stables of young horses), and the multiplicity of the cases, which may occur simultaneously in the same stable, are arguments favorable to a microbic theory. Dieckerhoff admits also, as an essential cause of the disease, the existence in the primary morbid centres of microorganisms that generate chemical poisons which have only a secondary action. These brief considerations show how obscure is the nature of petechial fever.

The manner in which the infectious matter is introduced is unknown. It is probable that it penetrates into the organism with inhaled air or ingested food.

Authors have observed in the ox a disease having characters similar to petechial fever of the horse, but none demonstrate the identity of the two affections.

Pathological anatomy. The principal alterations are hemorrhages of the skin, mucous membranes, subcutaneous and submucous tissues (all mucous membranes without exception show petechiæ), lungs, spleen, kidneys, red muscles, and myocardium. According to Zschokke, these hemorrhages are a consequence of mycotic endarteritis with thrombi and infarct; others think they are due, as in phosphorous poisoning, to fatty degeneration or a particular friability of the arterial walls; these lesions are produced by a specific agent. The most insignificant hemorrhagic centres are found upon the nasal mucous membrane, where frequently they are scarcely as large as a millet seed or a lentil; the largest, which are found in the lungs, are of the size of a man's fist. As consecutive alterations we remark an inflammatory tumefaction of the subcutaneous, submucous, and intermuscular connective tissues, or a gangrenous destruction of the affected organs (skin of the surface of flexion of articulations, intestinal, nasal, and pharyngeal mucous membranes). The lacerations produced by the blood in the internal organs may occasion fatal pleural or peritoneal hemorrhages; sometimes the lungs are the seat of a parenchymatous phlegmasia. A gangrenous destruction of the inflamed tissues may be accompanied by perforating peritonitis, pyemia, or septicemia. Let us examine successively the alterations which take place in the different organs.

1. The skin, which is tumefied, especially in the dependent regions, is studded with hemorrhages, the dimensions of which vary from the size of a pea to that of a silver dollar; if raised and spread, it presents a spotted aspect. Sometimes the surface of the tumefied regions is cracked, in other cases it presents ulcerated areas in which the muscles are exposed. The subcutaneous connective tissue is infiltrated by serum and blood.

2. The nasal mucous membrane is covered with irregular petechiæ from the dimensions of a lentil to that of a hazel-nut; they are often confluent and form bands or more or less large spots. Sometimes the entire pituitary is infiltrated with blood, especially on the

surface of the turbinated bones, and the nasal cavities are reduced to narrow cracks; in many cases it shows deep gangrenous ulcers; the nasal wall has several times been found perforated. Similar alterations exist on the surface of the laryngeal mucous membrane (œdema of the glottis). There the bloody infiltration locates preferably on the epiglottis and the lateral sinus. In the larynx and the pharynx we may also find ulcerations; very frequently the retro-pharyngeal lymphatic ganglions are tumefied or have formed abscesses. In the mouth the petechiæ are more rare; they are located, as a rule, on the inner surface of the lips, gums, and tongue; as in other places, they may be transformed into ulcerations. The gastric mucous membrane is marbled; some hemorrhages exist under that membrane, also under the serous membrane and in the thickness of the muscular tissue; very fine and disseminated, they give to the section a bluish-gray coloration. On the side of the intestine we observe in the mucous membrane, in the subserous and submucous connective tissue, bloody extravasations of very variable dimensions; the muscular tissue is overrun with punctiform ecchymoses; the mucous membrane, which is more or less tumefied, shows ulcerations of various dimensions, with areolated granular tissue in course of cicatrization; sometimes they involve all the layers and determine a gangrenous peritonitis. These alterations are most marked in the intestinal grêle, but they are also found in the cæcum, colon, and in the rectum. In these latter organs the hemorrhages are frequently arranged in bands. The intestinal contents are sometimes bloody. In some patients the conjunctiva and the vaginal mucous membrane are covered with hemorrhages.

3. All the muscular masses (pelvis, thigh, abdomen, chest, tongue, jaws) show circumscribed hemorrhagic infiltrations, which are of variable form and dimensions, of a dark brown or copper-brown color, and are often quite numerous. In most cases the muscular fibres, which are pale or clay-colored, are granular and undergo fatty degeneration; the hemorrhages give to the muscles a marbled aspect; the connective tissue is infiltrated with serum and blood; sometimes the muscular portions are gangrenous and almost detached. The myocardium is marked with similar alterations: numerous sub-epicardiac, sub-endocardiac, and interstitial ecchymoses; it has generally a reddish-gray or clay-like coloration. On the bones we find sub-periosteal hemorrhages.

4. The lungs are studded with hemorrhagic centres varying in size from that of a nut to a fist; they are found under the pleura and in the pulmonary parenchyma; if they become confluent, a diffuse hemorrhage may be produced. We observe, besides, an extended croupous pneumonia, and sometimes numerous foci that have undergone purulent or gangrenous transformation (pneumonia by foreign bodies). Pulmonary hypostasis and œdema are also lesions which are nearly constant.

5. In the spleen and the kidneys there exist extravasations of blood of variable dimensions; their opening into the peritoneum may result in fatal hemorrhage. The splanchnic cavities contain a bloody exudate, which is sometimes very abundant in the abdominal cavity.

6. The blood does not suffer any marked alteration as long as the disease develops without any complication. During life it does not contain any micro-organism; shortly after death we find therein the bacilli of malignant œdema, as likewise in all cases of asphyxia.

7. We recognize, besides, the anatomical picture of septicemia, of pyemia, or of asphyxia; lastly, in cases where petechial fever is secondary we discover vestiges of the original disease (strangles, etc.).

Symptoms. 1. Petechial fever is generally manifested by the appearance upon the mucous membranes of numerous dark red petechiæ; sometimes they are as insignificant as a flea-bite, then again they may attain the size of a pea or an acorn; they often become joined and form spots or bands of variable length. The nasal mucous membrane has a peculiar marbled appearance; it is more or less tumefied and allows the transudation of a yellowish serum; when the petechiæ are very numerous therein it may present a uniform reddish-blue coloration; in serious cases it becomes affected by gangrene or covered with ulcerations. The discharge is bloody and of bad aspect; breathing is very laborious, and the expired air has a fetid odor. The general condition sometimes becomes very rapidly aggravated; then, in the majority of cases, the disease ends in death. A petechial eruption may exist during the whole course of the affection.

2. Corresponding with the appearance of the petechiæ, or a few days later, the skin becomes tumefied; this symptom, which is by far the most evident, is often observed from the very first. Cuta-

neous tumefaction assumes two very different aspects; sometimes we observe circumscribed swellings which range in size from a ten-cent piece to a silver dollar; they are preferably located upon dependent regions (extremities, head, abdomen, sheath, chest); at times we recognize tumors similar to those produced by anthrax, and which from the onset are of the size of an apple or of a fist. These swellings, which are soft, œdematous, and but slightly painful, are hard and very sensitive (on the head), and are remarkable by their rapid extension and by their simultaneous appearance in different regions. Whatever may be their characters in the beginning, they become confluent, the obstruction extends toward the upper regions, the affected parts become deformed, the legs constitute cylindroid pillars, the head resembles that of a rhinoceros. The skin of the tumefied parts is often hairless; the supporting line of the muzzle is generally marked by a depression. The surfaces of flexion of the articulations are fissured and cracked; the skin becomes gangrenous and falls in shreds; there are also produced fetid ulcerations without any tendency toward healing. As long as the skin preserves its vitality it is warm and sensitive; sometimes small drops of serum ooze from it; as soon as death takes place it becomes cracked and is soon detached. This process may be extended even to the muscles. Independently of this spontaneous necrosis by anemia, the skin often shows other lesions which are caused by various external influences, especially by decubitus (supporting region of the halter and surcingle, hips, shoulders, eyebrow arches, etc.). A rapid disappearance of cutaneous tumors is a very unfavorable critical symptom; as a rule, it indicates a near and fatal termination. In certain cases it is the expression of a very abundant sero-hemorrhagic extravasation which takes place in the intestinal mucous membrane; in other instances it is produced by an intense hyperthermia, by exaggerated organic mutations which absorb all disposable material.

When the tumefactions are specially localized on the legs, which is the rule, they occasion troubles of locomotion: a stiff and heavy walk, lying or rising is impossible; there are also pains during flexions of the articulations; often the patients refuse to perform the slightest movement. In cases where tumefaction of the face is very great the nostrils become more and more contracted, and may close entirely; we recognize a growing dyspnœa, which ends in asphyxia if we do not interfere in time.

3. On the side of the digestive apparatus we remark ecchymoses and sometimes ulcerations of the buccal mucous membrane. The appetite may be preserved during the first days, when cutaneous tumefactions and petechiæ in the pituitary already exist. Later, mastication is rendered difficult or even impossible by infiltration of the lips and cheeks. With the fever, disturbances of appetite and constipation occur. Deglutition is frequently rendered impossible by alterations of the pharyngeal mucous membrane (blood suffusions and ulcerations), of the subjacent connective tissue (œdema); sometimes also by inflammatory tumefaction of retro-pharyngeal lymphatic ganglions. In these cases petechial fever is complicated by symptoms of pharyngitis: dysphagia, salivation, regurgitation, accumulation of fodder between the molar arcades and cheeks, extension of the head upon the neck, fetid odor of the mouth, tumefaction of the throat, etc.; ordinarily the condition of the patients becomes very rapidly aggravated. Colics, which occur sometimes during the course of the disease, constitute another unfavorable symptom; they are usually intermittent, and are produced by hemorrhages and inflammatory tumefaction of the intestinal mucous membrane. The dung is ordinarily coated with muco-purulent membranes, a phenomenon due to phlegmasia of the rectal mucous membrane; at the time of defecation we may see this mucous membrane tumefied and covered with petechiæ. When the lesions of the intestine determine its paralysis or the perforation of its coatings, they lead rapidly to death.¹ The urine sometimes contains a certain quantity of blood (hematuria). Micturition may be rendered difficult by tumefaction of the sheath.

4. If the temperature is ordinarily normal or hardly increased in the beginning, there are, however, cases where it reaches almost immediately 40° to 41° C. The petechiæ do not occasion any febrile reaction; later, when inflammation is added as a complication of these hemorrhages, fever appears and gradually increases, although remaining moderate (39.5° to 40° C.) in a great many patients. High temperatures indicate complications. The pulse, which is normal in the beginning, becomes accelerated when painful inflammatory tumefactions exist under the skin; we often count from 60 to 80 beats per minute; this acceleration is still more evi-

¹ Invagination is a possible complication of these intestinal lesions. Railliet has mentioned three observations of this nature. (See *Bullet. Soc. vét. et Archiv. vét.*, 1877.)—N. D. T.

dent when serious visceral alterations are produced. The number of pulsations is not at all in proportion to the temperature. The general condition is not alarming; however, when the disease is likely to end fatally weakness is ordinarily very pronounced, and we observe abundant sweating spells.

5. Tumefaction of the nostrils, inflammatory processes of the laryngeal mucous membrane and of the lungs, cause alarming respiratory complications. As soon as the opening of the nasal cavities and of the larynx is considerably reduced, we hear bruits of contraction, and asphyxia may occur; it is most frequently occasioned by œdema of the glottis. The symptoms produced by pulmonary lesions are sometimes but little marked and easily pass unobserved; however, in numerous cases hemoptysis is characterized by a bloody discharge, croupous pneumonia by dulness and bronchial wheezing, gangrenous pneumonia by a fetid odor of the expired air and by serious general symptoms; finally, pulmonary œdema by an increasing dyspnoea and by crepitant râles, which may be heard during auscultation.

6. Ocular manifestations are not rare: The conjunctiva, which is often overrun with hemorrhages, is tumefied and permanent; the lachrymal secretion is orange-colored or bloody. At the ophthalmoscopic examination we may find in the iris, the choroid, and the retina ecchymotic centres which are produced outside of any external influence. Röhl has several times observed a destruction of the eye by abundant hemorrhages. Schindelka has recognized atrophy of the optic nerve as a consecutive alteration.

Course. This is very irregular and atypic. There are benign cases in which an absorption of hemorrhagic and inflammatory centres takes place within a week without the occurrence of any serious ulterior phenomenon. In this slight form infiltrations and tumefaction of the skin persist ordinarily from eight to fourteen days; later a cure is produced. When hemorrhagic infiltration of the skin and mucous membranes is more marked, especially when extravasations exist in the pharyngeal and intestinal mucous membranes, they are only very slowly absorbed; the disease lasts from four to six weeks, sometimes longer. In a series of seventeen cases observed by Zschokke, the average duration was six days; the shortest was forty hours (fatal ending), the longest forty-three days.

Death may occur within a few days through internal hemorrhages, by septic infection, or by paralysis of the intestinal grêle.

This is almost always the usual ending in affections of gangrenous pneumonia (by foreign bodies). In cases where it takes place slowly the patients die of septicemia or of pyemia, after having shown the principal symptoms of these diseases: weakness, intense fever, mental depression, diarrhea, etc. Death is also the usual ending when petechial fever is associated with another serious disease. Cutaneous ulcerations become cicatrized, but very slowly; often they disappear only after several months. When they exist upon the surfaces of flexion of the articulations they may occasion cicatricial retractions (bonleture, ox-knee). There are cases where these final alterations are so serious that the animals must be killed after having been treated for many months.

Differential diagnosis. Strangles, anthrax, septicemia, pyemia, acute glanders, contagious pneumonia, certain poisonings (phosphoric and mercurial poisoning in the ox), may be accompanied by petechiæ of the nasal mucous membrane or by circumscribed tumefactions of the skin. In the beginning it is not always easy to distinguish these morbid conditions from petechial fever. But we may affirm the latter when numerous or diffuse cutaneous tumefactions and generalized petechiæ exist. These characters enable us also to differentiate it from urticaria and common phlegmonous diseases of the skin.

Prognosis. The long duration of petechial fever, the frequency and multiplicity of its complications, require a certain reserve in the prognosis. The mortality is about 50 per cent. Of seventeen patients which were treated by Zschokke twelve (70 per cent.) died. As fatal prognostic symptoms we may mention: numerous generalized petechiæ; diffuse inflammatory tumefaction and necrotic phenomena of the skin and mucous membranes; serious symptoms originating in the pharynx, larynx, and the intestinal mucous membrane, tumefaction of the nostrils, intense hyperthermia, very marked acceleration of the circulation (80 pulsations and more per minute), entire inappetence, rapid disappearance of cutaneous tumefactions, fetid odor of the expired air, very pronounced weakness, a decubital position, profuse diarrhea, stupefaction, general dulness, and the complication of pneumonia. The favorable prognostic symptoms are the localization of the petechiæ and the tumefactions, preservation of the appetite, and a normal temperature.

Treatment. The patient must be placed in a stall which is sufficiently large enough for it to move about freely. The halter

and surcingle should be removed. When dysphagia occurs we must give green fodder and slops.

A great many medicinal agents have been employed in combating the disease. Let us particularly mention: calomel, salicylic acid, sugar of lead, gallic acid, ergot of rye, iron preparations, camphor, quinine, arsenious acid, phenic acid, salicylate of soda, cresol, ichthyol (Lustig), essence of turpentine, sulphuric and hydrochloric acids. We generally use boric acid (20 grammes in drinking-water) and quinquina bark (50 grammes per day in electuary). All these remedies seem to exert a favorable action in some cases, but most of the time they produce no useful effect.

Dyspnœa which is of nasal origin may be combated by a suture which joins the wings of the nostrils, or by the introduction of metallic cylinders in these openings (Johne). But, as a rule, we must resort to tracheotomy, and it is important to practise it early.

[This operation is always attended with considerable risk, as it not infrequently happens that the wound becomes the seat of what is spoken of in this book as malignant œdema of Koch (septicæmia). My own experience with tracheotomy in purpura hæmorrhagica has been that animals in which the disease is so severe as to make this treatment necessary are animals that invariably die; but we have not done our whole duty to the owner of our patient if we neglect to do anything which offers a chance of success. The administration of three to four ounces of spirits of turpentine, together with one or two quarts of strong infusion of coffee daily, will probably give a larger percentage of cures than any other treatment yet proposed; certain conditions (as, for instance, abscess), when they arise, should be combated by specially indicated treatment. A very liberal supply of fresh air and perfect sanitary conditions are indispensable; diffusive stimulants and antipyretics are always indicated.—W. L. Z.]

The local treatment of ulcerations by means of astringents appears to us to be superfluous; we have tried Burow's solution without having obtained any favorable effects.¹ In order to diminish the sensitiveness, which is due to a tension of the tissues, we resort to applications of pure oil or paraffin ointment; irritating frictions are to be rejected in all cases. Superficial scarifications of greatly

¹ Burow's solution is composed of: alum, 5 parts; acetate of lead, 10 parts; distilled water, 500 parts.—N. D. T.

tumefied regions have appeared to Röhl as well as to ourselves to be frequently hurtful and never of any benefit. They were used formerly in order to prevent gangrene. Dieckerhoff insists, however, on the advantageous effects of deep incisions practised in tumors. The treatment of gangrenous and ulcerous alterations is purely surgical.

In these latter years Dieckerhoff has recommended Lugol's solution for combating petechial fever, administered in intra-tracheal injections. Every day he injected from 10 to 30 grammes of a solution of 1 per cent. (iodine, 1 part; iodide of potassium, 5 parts; distilled water, 100 parts). Adam, Gröning, Dobesch, Schröder, and others have mentioned a few instances of cures which were obtained by these injections.

Zschokke, who has used this treatment in a dozen cases, considers it as hurtful, and declares that it does not give anything but bad results. In the Dresden clinic three patients which were treated with iodine injections died; in two others iodine and calomel were used—the first recovered, the second died (Johné); another horse, which was treated by calomel only, recovered. In two cases iodine injections were followed by necrotic tracheitis and pulmonary gangrene. We have observed a case of granular tracheo-bronchitis, which was caused by iodine injections. Lemke and Buch have seen horses die after an injection of 30 grammes of Lugol's solution.

Petechial fever, if left to itself, ends in a cure in about half of the cases; in order to form an opinion as to the efficiency of the iodine treatment we must wait for a larger number of facts. At the present time its disadvantages appear to be greater than its advantages. Trinchera makes the same observation by reason of the results of the treatment of glanders. Practitioners must, therefore, take into account the serious accidents which this new therapeutic method may produce.

STRANGLES OF THE HORSE.

Etiology. Strangles of the horse is an acute infectious disease, which consists essentially in a catarrh of the mucous membrane of the anterior respiratory tracts, with suppurative inflammation of the lymphatic ganglions, which are traversed by lymph from the inflamed regions. The infectious agent—the streptococcus of Schütz—

exists in the pus of the inflamed glands ; it is easily shown by using aniline colors (gentian violet, etc.). It cannot be cultivated either in peptonized gelatine or in agar. In meat broths it forms at the bottom of the tube a flaky mass ; on coagulated mutton serum it produces transparent gelatinous drops, containing small grayish flaky masses which soon become confluent. This streptococcus is pathogenic for the mouse ; it produces an abscess at the place of inoculation and metastatic processes by way of the lymph or blood-vessels ; it is a specific and pyogenic micro-organism. These characters permit us to distinguish it from other *cocci* which are known at the present time, especially from the *Streptococcus pyogenes*, which may very easily be cultivated in peptonized gelatine and in agar. In the horse the cultures of the streptococcus of Schütz determine abscesses at the place of inoculation ; if carried into the nasal cavities, they generate strangles.

Jensen and Sand have also found in the pus of strangles a bead-string-shaped coccus (*Streptococcus equi*), with which they have produced infectious disease in the horse and mouse. They have succeeded in cultivating it upon peptonized gelatine and agar. Attempts at infection by inhalation of this microbe have remained without result. These authors think that infection does not take place through the intact mucous membrane.

Pathology. Strangles is a contagious, perhaps miasmatico-contagious, disease. According to Schütz, specific streptococci form arthrospores, which very probably exist outside of the organism, and which, under these conditions, may also be multiplied. Lafosse (1790), Viborg (1802), Erdelyi (1813), Toggia Reynal, and others have succeeded in transmitting strangles. The infectious agent resides especially in the secretions of the nasal mucous membrane, but it spreads in the air of infected stables. In the last century Erasmus Darwin attributed the disease to an atmospheric miasm.¹

Strangles is observed exclusively in the horse, donkey, mule, and hinny. It is sporadic, enzootic, or epizootic. While it most frequently affects young horses (from two to five years), it is also found in very old animals (from twenty to twenty-five years), as Jensen and Sand have again recently demonstrated ; it may also affect colts during the first weeks following birth ; in this respect it shows a certain analogy to the disease of young age. According to Jonsson, it is unknown in Iceland, a peculiarity which is ex-

¹ Simon : Die Drüse der Pferde, Erlangen, 1811.

plained by the specific and contagious nature of the disease. A first attack confers immunity for several years at least, and often for a lifetime. This immunity depends, perhaps, upon a supuration of the lymphatic ganglions, which destroys one of the centres of development which are most favorable for the infectious agents of strangles.

The predisposing causes are young age, a delicate or weakened constitution, cold, catarrhal diseases (which act by favoring infection), bad weather, changes of season in spring and autumn, want of condition, permanent stabling, excessive exertions, emigration, insufficient hygienic care, etc. Strangles may take on an enzootic form in crowded stables, in horse markets, in young horse depots, in the army, and on stud farms.

It is principally through the respiratory mucous membrane that the infectious agent produces general infection. Intense fever and very marked hyperthermia, which appear from the beginning, even before the development of ganglionic tumefactions, indicate that this agent passes directly into the blood. Experiments which have been made by Schütz, Jensen, and Sand have given results confirming this opinion. At a more advanced period the streptococci locate preferably in the lymphatic system, especially in the ganglions, where they produce abundant suppuration. After having traversed the capillary walls they may stray between the parenchymatous cells of corresponding organs without the intermediary of the blood and lymphatic ducts. It is very probable that in certain cases they penetrate through the digestive mucous membrane; the frequency of abscesses in the mesentery and in the submucous connective-tissue layer of the intestine, the alterations of Peyer's patches and of solitary follicles, do not leave any doubt to that effect. Frequent suppuration of the bronchial ganglions also indicates that they may invade the organism through the pulmonary mucous membrane.¹

The period of incubation of strangles is relatively short; its average duration is from four to eight days.

Old theories upon the nature of strangles have but a historical

¹ In countries where stock is raised, where strangles is common—almost permanent, contagion often occurs by accidental or operation wounds. Streptococci may be deposited therein by instruments, by the operator's hands, by assistants, grooming utensils, etc. (See Jouquan: *La Gourme de Castration*, Bull. Soc. Cent. Vét., 1888.)

interest. According to the period and the medical doctrines which existed, it has been considered as a disease of development, or like a disease *à frigore*; it has been related to a kind of lymphatic constitution which was said to be proper to the horse, to dental eruption, and to certain climatic conditions. Some authors have likened it to scrofula or to measles of man. Trasbot (like Sacco in former times) has identified it with *horsepox*; he advises to inoculate artificially all young horses with the lymph of the variola of the horse (Viborg and Toggia had recommended the same operation as a preventive of strangles). Delamotte has combated this theory, opposing it with experiments in which inoculation of *horsepox* has not protected the animals from strangles. Formerly several varieties were recognized: true strangles of colts, benign strangles, the suspicious or malignant, the chronic, migrating, transposed, aborted or returned, gangrenous, putrid, asthenic, composed and vesicular forms, etc. They believed in the existence of a specific virus which had to be eliminated from the organism, to a transformation of strangles into glanders, etc. Ancient authors designated it as laryngeal disease, goitre, adenitis, *morbus glandulosus equorum*.

Symptoms of normal and benign strangles. Like all infectious diseases, strangles begins with an intense fever. The general temperature rises from 40° to 41° C.; on the following days it drops from $\frac{1}{2}^{\circ}$ to 1° , only to rise again as soon as the lymphatic ganglions undergo purulent degeneration; when the pus is eliminated the temperature drops again. The pulse, which is normal, contrasts with hyperthermia. When in a quiet condition we count from 50 to 60 pulsations per minute; this figure is only exceeded in horses of a weak constitution or when complications occur.

The first local symptoms are those of acute catarrh of the nasal mucous membrane. The latter is uniformly red or covered with petechiæ; it secretes an exudate which is at first serous or viscous, and which becomes mucous from the third day; later, purulent and whitish-gray or greenish-yellow. This purulent catarrh, which is almost always bilateral, is sometimes more marked on one side than on the other. While in young animals the discharge is always very abundant, in aged horses it may hardly become apparent. In very benign cases, when strangles is *abortive*, the process retrocedes immediately and the ganglions do not form abscesses; but in the

great majority of patients purulent nasal catarrh is accompanied by a hot and very sensitive tumefaction of the ganglions of the tongue (submaxillary); the inflammation extends to the periganglionic connective tissue, the lymph accumulates in the afferent canals, an enormous obstruction occurs which at times fills the intermaxillary space and extends above the lower jaw upon the lower parts of the cheeks. As a rule the ganglions form abscesses; it is rare that the tumefaction is absorbed or indurated. Suppuration is marked by a fluctuation of the central part of the phlegmonous mass; the skin is distended, bluish, and covered with a lymphoid viscous exudate; soon the hair drops off, the integument becomes thinner and necrosed, a small hemispheric or conical swelling becomes apparent, and finally the abscess is opened and discharges a creamy pus, which is thick, yellowish-white, and sometimes mixed with shreds of necrosed tissue. Quite frequently the abscess opens at several points. The purulent cavity is filled by a swelling of its walls. After the spontaneous (eight to twelve days) or artificial opening of the abscess tumefaction and fever disappear rapidly.

Among the accessory symptoms we may mention a lessening of appetite or anorexia, which is due to a febrile state and to a tumefaction of the intermaxillary space, depression, muscular weakness, œdematous tumefactions of the posterior legs, and a more or less marked emaciation when the disease lasts a long time. Under the influence of fever the urine undergoes modifications; it has an alkaline reaction; it often contains a large proportion of albumin. During convalescence we sometimes observe a passing polyuria.

Finally, strangles may be accompanied by a cutaneous exanthema marked by swellings similar to those of urticaria (*Quaddel-*auschlag**) or, more rarely, by pimples, vesicles, or pustules, which appear upon the shoulders, the fascia of the neck and of the chest. This exanthema is remarkable in its development and its very rapid disappearance. On the pituitary, the skin of the nostrils, and upper lip we may also observe vesicles, the contents of which are limpid and watery in the beginning, but which become purulent later on; these vesicles burst, and are transformed into superficial ulcerations, which become covered by a thin layer and heal without leaving a cicatrix. It is to these fugacious lesions of the nasal mucous membrane that the name "phylyctenular strangles" was formerly given; but this denomination has also been applied

to cases of pustulo-contagious stomatitis, as well as to lymphatic processes, which will be described among the complications. *Urticariiform* eruption of the pituitary is much more rare than the preceding form; it is characterized by blackish sero-hemorrhagic infiltrations, which are of variable size, and are often surmounted by a red zone. As a general rule these exanthemata are quite rare. We may explain their appearance by a vaso-neurosis, by a passing vasomotor paralysis of circumscribed vascular regions of the skin and mucous membranes. Some individuals seem predisposed to it.

When strangles evolves in a normal way and is free from any complication, its average duration is from two to three weeks, and its ending is nearly always favorable.

Complications. 1. One of the most frequent complications is the extension of infectious catarrh to the pharyngeal mucous membrane; there are even cases where the affection begins by a phlegmasia of this tissue. The symptoms of pharyngitis predominate; we observe dysphagia, ptyalism, and regurgitations. The retro-pharyngeal lymphatic ganglions (subparotid) become inflamed and suppurate; these alterations are marked by a diffuse tumefaction of the lower parotid region; the ganglions which are situated on the base of the auricular pavilion between the parotid lobules (posterior maxillary ganglions) may participate in the inflammation. Suppuration is the ordinary termination of this adenitis; in some cases the pus has a tendency to discharge externally; in other instances it progresses internally toward the larynx and the pharynx; it may undergo caseous transformation after a partial absorption; it may also suffer putrid decomposition and ultimately produce septicemia or pyemia. By their confluence the abscesses often form vast purulent collections. Sometimes the submucous and peri-pharyngeal connective tissue, as well as the muscular surfaces of the region, is overrun with small abscesses. From the retro-pharyngeal ganglions the suppurative inflammation is sometimes radiated to a great extent by progressing in the subcutaneous connective tissue and producing enormous tumefactions of the head and shoulders. The pus of the ganglions which have formed abscesses may work its way out, either directly or by settling toward dependent parts; this is the most favorable termination. There are cases where it penetrates into the larynx and pharynx after having necrosed the mucous membrane of these organs; in such cases gangrenous pneumonia is a frequent com-

plication ; there are others where it works through from inside and outside at the same time, and thus determines a guttural or pharyngeal fistula ; it also happens, but much more rarely, that abscesses of the submaxillary ganglions open in the mouth and produce a buccal fistula.

2. Infectious inflammation may be communicated to the mucous membrane of the larynx, trachea, bronchi, accessory cavities of the nose, buccal cavity, guttural pouch, and to the conjunctiva, etc. A laryngeal phlegmasia, which is at first catarrhal and superficial, is marked by frequent coughing ; quite often it extends in depth, becomes phlegmonous, and determines a serious dyspnœa ; sub-mucous abscesses, inflammation, paralysis of the laryngeal muscles, and wheezing (Friebel and others) sometimes occur during the course of this laryngitis. Catarrh of the bronchi, as a rule, produces tumefaction and suppuration of the bronchial ganglions. It is also possible that the walls of the guttural pouches may be affected by partial necrosis ; then alimentary matters penetrate into it and become fixed in the peritracheal connective tissue. However, in a general way, strangles of the guttural pouch are rare.

3. Superficial lymphangitis of the head, accompanied by numerous subcutaneous abscesses, constitutes another special complication of strangles. It is especially found upon the lips, cheeks, wings of the nose, and even over the whole surface of the head as far as the ear and beginning of the neck. The numerous lymphatics of the skin are inflamed ; the surrounding connective tissue is infiltrated with pus. This perilymphatic suppuration produces small abscesses arranged in lineal series like a string of beads along the course of the bloodvessels, and which are altogether independent of suppurating adenitis. Lymphangitis leads to tumefaction, tension of the lips and cheeks, the mucous membrane of which is indurated ; the disseminated glands in the latter may also suppurate and form follicular adenitis, from which purulent masses escape when the lips are turned up, and which are transformed into small ulcerated wounds.

4. Another constant and quite remarkable fact is the tendency to metastases in the different organs (pyemic strangles of Schütz). These metastases are produced in some cases in the ganglions of the neighborhood through the medium of the lymphatics ; in other instances, in distant organs, in the different viscera, through the

blood (emboli). They consist of purulent collections of variable size, accompanied by phlegmasic phenomena which are more or less intense. They may be found in the neighborhood of the thyroid glands immediately in front of the trachea; in the medium-sized cervical ganglions behind the trachea [when tumefaction exists on the left side toward the middle of the jugular gutter it determines in some cases chronic wheezing (Fiedler); the abscess may open at the same time upon both sides of the neck]; in the sterno-hyoidean and omohyoidean muscles; in the lower cervical ganglions (tumefaction at the level of the entrance of the trachea into the chest; at times it depresses this canal and determines asphyxia); in the axillary ganglions (lameness); under the skin of the pectoral walls, of the chest, hypochondria, belly, flank, scrotum, mammae, neck, withers, and of the inner fascia of the posterior legs, etc. Subcutaneous abscesses produce, in the region where they are developed, an enormous and diffuse swelling; they are often taken for common oedematous tumefactions of the integument. In cases where the bronchial ganglions are overrun by suppuration we find mediastinal abscesses from the size of a pea to that of a man's fist, and sometimes empyema due to the penetration of pus into the pleural pouches. Similar purulent collections are found in the omentum, the mesentery, the connective tissue of the pelvis, in the mesenteric ganglions, the pancreas, kidneys, etc.; they may contain as much as ten litres of pus; their opening through the abdominal serous membrane produces a fatal peritonitis; during life they cause slight periodical colics, which are sometimes prolonged for years. In the intestine we may recognize submucous abscesses, tumefaction and suppuration of the closed follicles, alterations which are also marked by remittent colics. Suppurative lesions exist more rarely in the brain (symptoms of encephalitis, immobility, blindness), in the spinal cord (paraplegia, paralysis of the rectum and of the bladder), in the articulations [knee, hock, boulet, coronet (symptoms of suppurative arthritis, tumefaction, functional troubles)], in the mammae (purulent mastitis), in the lymphatic ganglions of the extremities, especially in the popliteal ganglions. Abscesses which develop in these latter organs determine lameness of long duration and emaciation of the muscular masses of the hip; often the tumefaction which betrays them only becomes visible after a very long time, because the ganglions are situated deeply, under the bifemoro-calcanean muscle;

when these abscesses are not opened in time the pus finds its way toward the inner fascia of the member. The disorders which are produced by it may occasion persistent lameness.¹

5. The pulmonary alterations are those of metastatic pneumonia, by foreign bodies or simply catarrhal. In most cases pneumonia by foreign bodies is consecutive to pharyngitis, and is produced by a penetration into the respiratory tracts of the pus of peripharyngeal abscesses.

6. Strangles takes a chronic type when the process is localized in the maxillary and frontal sinus, in the pharynx, or in the guttural pouch. Fever and discharge persist, the general nutrition becomes impaired, digestive troubles appear. We may observe phenomena which cause a suspicion of the patients being affected by glanders (suspicious strangles).

7. A fatal ending is ordinarily the result of septicemia or pyemia.² In these cases fever is very high, the pulse is accelerated and weak, the heart-action palpitating, the general sensitiveness much dulled; later, diarrhea occurs, and often the depression of strength appears with extraordinary rapidity. Frequently, but especially at the period of convalescence, petechial fever complicates strangles.³

¹ Abscesses which develop in the withers ("symptomatic disease of the withers") are often accompanied by serious lesions, by purulent spurts between the muscular planes, under the shoulders, or by necrosis of the hard organs of the region, especially of the dorsal spinous processes. They are marked by a diffuse, hot, and very painful tumefaction of the region; sometimes it is bilateral, in other cases it exists on one side only. In proportion as it increases we observe an attenuation of the symptoms of strangles (Trélut). Incisions which are made into this phlegmosed tissue (opening of abscesses) occasion abundant hemorrhages. This complication of strangles is not the result of external irritations acting upon the withers (action of the collar, saddle, wound, contusion); it has been often observed in fillies and broodmares without any intervention of these causes. (See Trélut: *Revue Vét.*, 1883.)—N. D. T.

² In a case reported by Decoste death was caused by an opening into the rachidian canal of an abscess which was developed in the ilio-spinal (see *Receuil Vét.*, 1834). In another, which was related by Chauvrat, the animal died of a similar complication; the abscess was situated on the lower fascia of the atlas and axis, under the long muscle of the neck, the anterior straight lateral, and the great oblique of the head. (See *Receuil Vét.*, 1885.)—N. D. T.

³ Strangles may be transmitted from the mother to the fœtus through the placenta and cord. Chorsy found pulmonary, intestinal, and ganglionic lesions in a filly that had died a few minutes after its birth, the mother of which had been affected with strangles during the eighth month of gestation. Nocard found isolated micrococci doubled or associated in small chains which were identical with Schütz's streptococci. (See *Bullet. Soc. centr. Vét.*, 1888.) Nocard and Wiert, and quite recently Mègnin, have reported two other similar facts (*Ibid.*, 1890-91).—N. D. T.

[Subparotid abscess, often appearing as a secondary invasion of this disease, is one of the most common complications occurring in general practice, and especially in horses past the adult age. It is not uncommon, after having treated and discharged as cured a case of simple strangles, to be called a week or ten days later and find a subparotid abscess, which is not infrequently complicated with an ophthalmia or a panophthalmitis. Psoas abscess is sometimes seen in weanlings; this abscess will open by means of fistulous tracts in the hypochondriac region following the course of the ribs in the perineal region, following the rectum in the loose connective tissue of the pelvis and in the groin. Convalescence in these cases may be prolonged several months, but is usually complete.—W. L. Z.]

Differential diagnosis. Strangles may be confounded with several diseases, especially with common catarrh of the nasal mucous membrane, which is also marked by a muco-purulent discharge. Though quite common in young animals, this catarrh may, however, be distinguished from strangles, as it is never accompanied by purulent phlegmasia of the submaxillary ganglions. In certain cases of common subacute catarrh we may, it is true, recognize a slight tumefaction of the ganglions, but then these latter never form abscesses. (For a differentiation from glanders see Differential Diagnosis of Glanders.)

Prognosis. It is generally favorable; out of 100 patients only a few die. A statistic of Ringheim established that out of a total of 2205 horses which were affected by strangles, and had been treated in the Danish army from 1851 to 1860, a mortality of but 0.5 per cent. was recorded, a very small proportion, which is explained by the advanced age of the animals (four to nine years). Krabbe, in calculating 1789 cases of horses of all ages which were affected by strangles, has found it to be 1.6 per cent.; another statistic, due to the same author and referring to 1107 horses, has given a proportion of 3.5 per cent. In Denmark in 1885, out of a total of 2419 horses, the loss was 2.3 per cent.; in 1883, out of 2381 patients, it was 2.6 per cent. (Goldschmit). The average rate of mortality from strangles is thus 2 to 3 per cent. The strong constitution of the subjects and the regular course of the disease are favorable conditions; on the contrary, an inherent debility, or that which is produced by previous diseases, young age, various complications, is against the prognosis.

Treatment. In cases where the disease evolves in a normal

manner it is sufficient to subject the patients to a diet, to keep them clean, thoroughly ventilate the stables, and give food which is easily digested (green fodder, roots, slops). In the beginning it is useless to resort to antipyretics, for the febrile phenomena disappear within a few days. The treatment of suppurating adenitis is purely surgical. As soon as the abscesses are recognized they should be opened with a free incision. In proceeding thus we decrease the fever and the duration of the disease is shortened. When purulent absorption of the inflamed ganglions takes place slowly we may try to hasten it by means of Priessnitz's compresses (with phenicated or cresol water), or by means of vesicant frictions. Puncture of the abscesses of the parotid region must be made with the necessary precautions in order to avoid the numerous blood-vessels of that region; as a rule, the skin alone is cut by means of a bistoury, and the track leading to the abscess is made with the finger. If we do not reach the purulent collection at the first attempt, these manœuvres must be repeated upon another point, or we may resort to exploratory punctures made by means of a fine trocar.

Formerly we combated catarrhal diseases of the mucous membranes with fumigations of tepid water and with antimonial preparations (black and yellow sulphate of antimony), alkalies (sulphate of soda, hydrochlorate of ammonia, bicarbonate of soda, etc.), or sweet substances (licorice, honey). When the catarrhal processes are obstinate we administer artificial Carlsbad salts: sulphate of soda, 75 grammes; chloride of sodium, 20 grammes; bicarbonate of soda, 5 grammes—a tablespoonful at each meal. We may also prepare an electuary with: sulphate of soda, 250 grammes; black sulphate of antimony, 25 grammes; licorice and marshmallow powder—to be administered twice a day. A number of medicamentous additions have been recommended. Most of the powders used to combat this disease (proprietary), and which are much liked by the public, contain a certain proportion of the agents which we have just mentioned, or some similar products, but they are especially composed of tainted vegetable powders, and often they do more harm than good. Träger, more than half a century ago, pointed out their defects and condemned their use. All classical treatises have repeated this criticism in vain.

Complications require special treatment. Dyspnœa may necessitate tracheotomy. The prophylaxis requires isolation; the sick animals should be separated from healthy ones.

Different authors have recommended to inoculate the disease in healthy animals in order to give them immunity by means of a benign attack. Jensen's and Saud's experiments show that intravenous injections of the micrococci of strangles do not produce general infection, but only acute phlegmons, which create immunity against ulterior infection of the nasal mucous membrane. Nevertheless, in this respect it is advisable to wait for new experiments. It is probable that we shall not decide so quickly, at least in Germany, to practise the method of artificial infection recommended by Peterson. This author has subjected all the colts of a stock farm to the action of cold: one day during autumn he had them enter a pond, where they were kept for half an hour; then he exposed all to cold winds upon high ground, and gave the same animals very cold water to drink. All of these colts were affected by strangles, and everyone was cured within three weeks.

DISEASES OF DOGS: CANINE DISTEMPER.

HISTORY. Canine distemper has at all times been considered a very fatal disease. Various names have been given to it: *epizootic of dogs*, *canine plague*, *canine glanders*, *canine disease*, *misery* or *weakness*, *catarrhal fever*, etc. Laosson enumerates 105 authors who have made a special study of it.¹ According to this veterinarian, it was already known in the time of Aristotle, and the epizootic which decimated the canine species in Bohemia during the year 1028 was canine distemper. However, up to modern times it has been generally admitted that the trouble was of American origin, and that it had been imported into Europe shortly after the discovery of the new world (according to Hensinger, it came from Peru). It is said to have first appeared in Spain, and from thence to have passed into France, Germauy, and other countries of the old world. Its appearance in France dates about 1740, in Germany from 1748, in Italy in 1764, in England toward 1760, and in Russia about 1770. At the present time it exists in all Europe.

Dissenting opinions have been expressed on the subject of its nature. It has been considered successively as a disease which is similar to the plague or to strangles, as a neuropathy, a mucous or

¹ Laosson : Thèse de doctorat, Dorpat, 1882.

nervous fever, etc. Some authors have likened it to typhoid fever, or have even identified it with typhus of man. A large number of others, and quite recently Trasbot, have considered it as a true variola; but the attempts which were made with the object of conferring immunity to young animals by inoculation of cowpox have been unsuccessful. Dupuy has just opposed this doctrine with new experimental results; never, in young dogs, has he obtained immunity by the inoculation of vaccine. We have also seen in canine distemper a disease which was connected with developement, a constitutional morbid state; it has been supposed that it was due to the insufficiency of soda salts in the organism.

However, its contagiousness and propagation by an infectious agent have been admitted for a long time (Waldinger, van Gemeren, Delabère-Blaine, etc.). They have also made numerous attempts at transmission with the morbid secretions. Renner and Karle have made known the first positive results obtained by inoculation. Trastowo has recognized that the disease is contagious for young dogs in which a first affection has not conferred immunity, that its transmission may be made directly or indirectly, and that sometimes old animals are affected. Inoculations which were made by Trasbot did not at first give any positive results; but afterward, when practised upon animals which were from thirteen days to three and a half months old, they invariably succeeded; they consisted in making slight scarifications upon the skin of the belly and covering these with a mixture of the discharge and product of secretion of the pustules. The symptoms appeared at the end of four or five days; within one week pustulation was complete. Trasbot has also demonstrated transmission by cohabitation. Venuta has confirmed this latter mode of contamination; he has established that the germ is "fixed" and "volatile" (in suspension in the atmosphere), and that it possesses sufficient resistance to undergo desiccation in the air without losing its virulence. According to this author, the duration of the period of incubation varies from four to six days. Krajewski has inoculated thirty-six animals. The result has been negative in the largest number, a fact which the author explains by acquired immunity. The period of incubation varied from four to seven days; the disease was invariably manifested by a rise of temperature. Exanthema was only observed upon a few animals which were seriously affected. In benign cases recovery occurred within six to eight days. Immunity has gener-

ally been conferred by a former affection. The germ was found in the discharge, in the ocular secretion, and in the blood. Desiccation, congelation, exposure to a temperature of 20° C., have not destroyed it; but if preserved in a dry state its virulence is weakened in the course of time. Krajewski has recommended inoculation as a prophylactic measure; the experimental disease gives a mortality of only ten to fifteen per cent. Laosson has made ninety-eight inoculations upon the dog and the cat; the results which were obtained have confirmed the contagious nature of the disease and established its identity in the canine and feline species; the disease of the dog is transmitted to the cat, and reciprocally. The virus has appeared to be *fixed* and *volatile*; almost all young animals were contaminated; many aged animals resisted the trouble. The discharge lost its virulence within a fortnight; the contents of the pustules appeared inactive. The period of incubation was from four to seven days. The degree of immunity which was conferred by a first affection varied according to the cases. Friedberger has succeeded in transmitting the disease by inoculating, by means of superficial punctures, the contents of the pustules into the derma of the internal fascia of one of the posterior members; the experimental disease was remarkable by its very short period of incubation (four days), also by its slight intensity, its rapid course, and by a pustular eruption which was localized upon the region where inoculation had been practised. Attempts at transmission which were made by Konhäuser have all given a negative result. According to this author, in nursing female dogs the contagious germ exists in the milk as well as in the product of morbid secretions.

Etiology. Canine distemper is an infectious and contagious pathological state the specific agent of which has yet to be determined. Its culture has not been successful, and consequently its transmission by isolated virus could not be effected. But it is known that it appears in *fixed* and *volatile* states. It is much more easily transmitted by cohabitation than by inoculation; the air seems to be the most common contagious medium. The period of incubation lasts from four to seven days.

Young animals are generally affected in the course of their first year. Out of 1378 patients which were treated at the Vienna school within one year, 927 (about two-thirds) were less than one year old, 269 (one-fifth) were from one to three years old, and 182 (one-seventh) more than three years. The disease is sometimes

observed in old dogs, as well as in very young animals (only a few weeks old). We have seen it in a whole litter of puppies during the third week following their birth. A first affection produces immunity for an indefinite period ; but this rule is not without a few exceptions.

As predisposing causes, the disease has been attributed to cold (want of shelter during the night, cold or damp weather, washing, baths, etc.), which seems to favor a penetration of the infectious agent, either by weakening the constitution, or in determining common catarrh of the respiratory mucous membrane. But cold of itself is not able to generate the disease. Weak animals, those having little resistance, also subjects of improved breeds, and such as have been recently imported and which are not yet acclimated, furnish a large contingent to this disease. An unsuitable regimen and defective breeding contribute to increasing the number of its victims. An old prejudice, which is still common, consists of a suppression of meat in the food of young dogs, and exerts an incontestable etiological influence, as a vegetable regimen determines in time a weakening of the organism of the carnivorous animal. In the experiments of Bischoff and Voit, dogs which were fed exclusively on bread became cachectic and the cats died. A weak constitution of the mother and an insufficient nursing when the young are numerous, render the progeny weak and non-resistant. Rha-chitis, abundant hemorrhages after the amputation of the tail and the ears, predispose also to the disease. The hypothesis that dogs of certain breeds are particularly subject to it, which is accepted by a large number of authors, is illusory ; it has very probably originated from the numerical predominance of these breeds. In Berlin, for instance, more than one-third of the affected dogs are of the King Charles variety, because this breed is very common there at the present time (out of 623 dogs which were treated at the Berlin school during the summer course of 1886 we counted 220 King Charles and but five grayhounds).

Bacteriology. The infectious agent of canine distemper is unknown. The assertions of authors who have investigated it do not agree. In the blood of diseased dogs Semmer and Laosson have found very thin and short bacilli, which they consider as the specific elements of the disease. Rabe looks upon these as schizomycetes which he has found in the contents of the pustules, in the discharge, and in the products of secretion of the conjunctiva.

They appear in the form of very small-sized corpuscles, which are at times grouped in an irregular mass, or which are sometimes associated in pairs or four by four (in the same manner as sarcina), in other instances yet disposed like a string of beads; they may be colored by means of methyl-violet. Their abundance in the secretions of the nasal mucous membrane is proportionate to the intensity of the process; they are no longer found during the period of convalescence. Friedberger has observed the micro-organism which is described by Rabe, but he admits only under reserve the rôle which is assigned to it by this author. Krajewski has found micrococci. Mathis found a specific diplococcus in the vitiated fluid tissues, discharges, and pustules. He has cultivated it in neutralized bouillon or alkali, and has obtained pure cultures (he made cultures in series and obtained seven reproductions of these). Inoculation of the latter has given positive results; the symptoms of the experimental affection were similar to those of the disease which is contracted in a natural way. In general, hyperthermia occurred very rapidly and pustules appeared on the points of inoculation and over the whole surface of the body. Most of the very young animals died; the surviving subjects had acquired immunity.¹

AFFECTED ANIMALS. The disease is observed in the dog, cat, fox, wolf, hyena, prairie dog, and the monkey (Schmidt and personal observations). In the canine species it exists in a sporadic, enzootic, or epizootic state. In large cities, where it exists permanently, the number of cases noticed varies according to the years; but, with the exception of simple gastro-intestinal catarrhs, it is the most frequent affection of the canine species. Out of 9000 dogs which were treated at the Berlin school during the years of 1886 and 1888, 3000 (33 per cent.) were affected by it. At the Munich school within fourteen years they have observed 655 cases in a total of 2320 patients (28 per cent.). The disease is also found in the country. In Iceland it has raged with such intensity that not one dog could be found within a large territory. Our obser-

¹ New bacteriological researches have been made at Nancy by Jacquot and Legrain. In the liquid of the pustules these authors have recognized numerous micrococci of 0.6μ to 0.8μ in diameter, mostly associated in pairs and apparently immobile. They succeeded in cultivating these in different mediums. Twenty young dogs were inoculated at different times with these cultures. Most of these showed a slight eruption at the point of inoculation; later they were not affected by the disease (see *Recueil Vét.*, 1890).—N. D. T.

vations establish that it is particularly frequent during the summer; heat seems to exercise a very favorable influence upon the development of the contagious germ.

Symptoms. The disease of canine distemper is marked by numerous complex manifestations; it consists essentially of an infectious catarrhal phlegmasia of the ocular, respiratory, and digestive mucous membranes; in many cases it is accompanied by serious nervous symptoms (cerebral and medullary), and also by a characteristic cutaneous exanthema; very often also it is complicated by catarrhal pneumonia. The diversity of the phenomena by which it is marked has led to division into several clinical varieties. According to the location of the morbid process on the mucous membranes, the central nervous system, or the skin, we distinguish the catarrhal, nervous, or exanthematous forms; ocular, gastric, and pulmonary forms have also been described. In fact, disease of dogs may be expressed by troubles which are simple as to their origin, by symptoms which are exclusively cerebral, intestinal, ocular, or cutaneous; but very often these troubles are multiple, and they may be associated diversely. It would not be possible to give a general typical description of the disease; we must limit ourselves in order to study successively its different localizations.

1. **SYMPTOMS OF THE BEGINNING.** In its initial stage the disease is marked, as a rule, by general phenomena: depression, a low-spirited condition, a capricious appetite or anorexia, fatigue, trembling, chills, erected hair, a dry and hot nose. In his researches Krajewski has noted an intense hyperthermia (up to 40°) toward the end of the period of incubation; at the time of the eruption the thermometer marked 41° C. in benign cases, 42° in serious cases. However, in a first examination, the fever is often moderate, and in slight cases, especially in adult and strong animals, the process is apt to evolve without a marked increase of the temperature. Thermometric examination can rarely be made in the early stage of the disease; the patients generally are not presented to the veterinarian until a more advanced period. But, as a general rule, canine distemper, like all infectious processes, begins with an ephemeral thermic elevation.

2. **OCULAR SYMPTOMS.** In a great majority of cases it is in the eyes that we observe the first local symptoms; they consist of a serous conjunctivitis which later becomes purulent; the eye is

watery ; there is photophobia, the eyelids are injected, the conjunctiva is red and tumefied. The exudate, which at first is mucous, then purulent, accumulates in the lachrymal pouch and in the inner angle of the eye, forming there small viscons, caseous, grayish, or yellowish masses ; it soils the edge of the eyelids, dries up during the day, covering these with crusts, and agglutinates them during the night. In the conjunctiva the purulent secretion is frequently blennorrhic. The irritation of the cornea by the accumulation and decomposition of the exudate, repeated scratching upon it by the patients, nutritive troubles which follow, produce upon this membrane true ulcerations. The epithelium is destroyed ; sometimes the losses of substance are great and superficial ; in other cases, which are more frequent, they are deep, infundibuliform, and of the size of a pin's head, and are usually located in the centre of the organ ; their bottom is often covered with a puriform exudate. In some cases the ulcerations heal through a vascular reformation which starts from their edge, in other instances they are extended to Descemet's membrane, the cornea becomes entirely perforated, the iris forms a hernia, and a staphyloma is developed. The terminations of the process are cicatrization, with persistence of white spots (leucoma), or by a dark pigmentation of the cornea, or purulent panophthalmia. In other cases we observe in both eyes a diffuse parenchymatous keratitis, which is characterized by a milky trouble of the cornea, which is sometimes developed within a very short time ; the surface of the cornea is smooth, shiny, and the conjunctiva intact. This lesion and a fever which is of variable intensity quite frequently constitute the only symptoms of the disease (affection of the eye). It is very rare to observe an exudative inflammation of the iris with formation of a fibrinous or purulent exudate in the anterior chamber of the eye (internal ophthalmia).

3. SYMPTOMS OF THE DIGESTIVE APPARATUS. The principal are : modifications of the appetite, vomiting, redness and dryness of the buccal mucous membrane, intense thirst, infrequent defecation or fetid diarrhœa, which is mucous, frothy, or hemorrhagic (intestinal hemorrhagic catarrh). Icterus occurs only in exceptional cases. In weak animals and in advanced stages of the trouble the urine, which is often albuminous, contains coloring-matters of the bile. In a certain number of cases these are the only manifestations of the disease.

4. SYMPTOMS OF THE RESPIRATORY APPARATUS. All at once

we notice the symptoms of a catarrh of the pituitary: a serous, mucous, or purulent discharge, sneezing, and a nasal pruritus, which the patients try to allay by rubbing the nose with the paws. As a rule, both nostrils discharge a purulent matter of a yellowish-gray or greenish-gray color, mixed with bloody striæ; later the discharge may become fetid, putrid, and gangrenous; the nose is very often dry and cracked; often also the pituitary is covered by true ulcerations. The abundance of the discharge indicates the extension of the process to the turbinated bones and sinuses.

Accompanying rhinitis we find quite often a laryngeal catarrh which is marked by a cough, at first dry and hard, afterward moist, and accompanied by a discharge; this cough often produces vomiting through reflex action. From the larynx the phlegmasiæ may radiate to the trachea and bronchi. Bronchitis is indicated by an augmentation in the number of respirations, by a coarse vesicular murmur, and by râles. In bronchiolitis, which is frequent, breathing is much accelerated and dyspnoëic; a weak, painful cough is heard at the time of percussion of the thorax when the animals rise or when they are taken out of their stable; we perceive also dry or damp râles, with fine or coarse bubbles. In young and weak animals which do not succeed in expectorating the bronchial exudates the latter are drawn into the alveoli, and catarrhal pneumonia is developed. The principal symptoms are: a rise of temperature, dyspnoëa (buccal respiration), attenuation or disappearance of the vesicular murmur in circumscribed regions of the thorax, zones of dullness which are disseminated in both pulmonary lobes (when the inflammatory centres are located very deeply this symptom may be wanting), a sound of percussion, which at times is tympanitic and in some cases bronchial wheezing. The cough is weak and laborious; the discharge is often fetid. When cardiac paralysis becomes imminent we may observe symptoms of pulmonary œdema, intense dyspnoëa, crepitant râles with big bubbles, a tympanitic sound of percussion, a slow and regular lessening of the number of respirations.

5. NERVOUS SYMPTOMS. In weak and anemic subjects serious cerebral symptoms predominate, indicated by great depression and stupefaction; in robust animals we notice especially symptoms of active cerebral hyperemia: fiery eye, hot cranium, excitement, restlessness, rabiform manifestations; later, these troubles are followed by others which are produced by cerebral compression (stupefaction,

extension of the head). These phenomena characterize the nervous form of the disease. In this variety, which is quite frequent, we recognize also tonic or clonic contractions, sometimes generalized, and at other times localized in certain regions, and which are due to an increase of the reflex sensitiveness of the brain and spinal cord. The convulsions are often limited to one or several members, the movements of which may be similar to that of a pendulum for whole days, or to the muscles of the head, especially to those which are innervated by the facial or the motor branch of the fifth pair, or to the lips, cheeks, eyelids, to the muscles of the temples and masseters; in the latter case the jaws at times perform alternate movements of opening and closing, which seem automatic. We may also observe spasmodic contractions of the panniculus carnosus, especially in the region of the back. At certain moments violent epileptiform spasms appear, which may be either partial or general. The animals, which are anxious or overexcited, shake the head, wander without aim, or run around as if lost; the jaws are convulsively agitated, a frothy saliva runs from the mouth, the eye is haggard, the head and neck are thrown backward or carried laterally, the muscles of the face are spasmodically contracted. We may recognize epileptiform attacks: the subjects fall, uttering cries, they lose consciousness and undergo generalized tonic or clonic spasms, the rectal and vesical sphincters are paralyzed, there is expulsion of urine and fecal matter. Within a very short time (a half-minute to a minute) consciousness returns, and they again rise; for a certain time they remain weak and stagger. In some cases these epileptiform attacks are followed by permanent coma; we remark, though more rarely, ring movements, rolling, or convulsions of the extremities. These phenomena are due to arterial anemia and to venous hyperemia of certain regions of the cerebro-spinal centre, as well as to the action of infectious poison on the nervous elements of these regions.

Besides these spasms we observe paralysis. These two kinds of phenomena exist at times simultaneously, but, in general, paralysis follows slowly; it may be localized in certain muscular groups; it frequently extends to the hind quarters or even the whole body; then paresis of the motor nerves is marked by extreme muscular weakness. The animals stagger, their hind quarters sink; they often stumble or tremble on the four legs; there are some which can no longer remain standing. In a few cases the disease leaves after it

a permanent paresis of the hind quarters and a vesico-rectal paralysis. We have several times noticed paralysis of the tongue; this organ was flabby, hung out of the mouth, and prehension of food was extremely difficult. During the course of the disease we may also find deafness, amaurosis, cataract (very rare), loss of smell and memory, and even complete disappearance of intelligence (internal hydrocephalus).

6. SYMPTOMS FURNISHED BY THE SKIN. In nearly half of the cases a peculiar pustular exanthema is developed upon the inner fascia of the legs and on the abdomen. When the disease does not run its entire course, this exanthema may constitute its only symptom. On the surface of the skin very small red spots appear which, within twenty-four hours, are transformed into miliary pustules surrounded by a red zone (vesicular and pustular state). These pustules, which are of the size of a lentil, pea, or bean, dry up, forming a brownish-yellow crust, or they burst and create moist wounds. Healing takes place in six to eight days, preceded by an epithelial desquamation: the spots which were covered by pustules remain for some time marked with pale or rosy blotches. Very often the eruption is slight, discrete, and circumscribed to few regions; it may extend to the whole surface of the body and simulate impetiginous eczema, and become communicated to the integument of the external auditory meatus, or, but more rarely, to the buccal and ocular mucous membranes. Then the patients give off a fetid odor, and the skin becomes partially depiled; however, contrary to what we observe in sarcoptic mange, the eruption produces only an insignificant pruritus. This exanthema is sometimes accompanied by an acute preputial catarrh. Lastly, in a few cases we observe a generalized urticariform eruption.

7. GENERAL SYMPTOMS. We have stated that the temperature rises in the beginning; it increases also when localizations are produced (lungs, intestines, etc.); at the approach of death it often drops considerably below the normal (36° , 34° , even 32° C.). But in general the course of the fever is irregular and atypic. When the duration of the disease is prolonged emaciation appears and becomes more and more marked, the abdomen becomes tucked up, the ribs prominent, the hair dull and bristly, the orbits sunk, the mucous membranes pale; the gait is staggering and weakness is extreme. The patients give off a fetid odor; they are almost always found recumbent and under the influence of deep coma.

Course and Prognosis. If considered from the standpoint of its course, canine distemper offers a greater variety of modifications than any other disease of the dog. When it is marked by a simple pustular exanthema, by a parenchymatous keratitis, by slightly-marked nervous troubles, by a benign catarrhal phlegmasia of the respiratory or digestive mucous membranes, it has often a rapid, abortive course, and may end in a cure within eight to ten days. In most instances its duration is from three to four weeks. In cases of serious encephalic or medullary complications the disease takes a slow course and is followed by several morbid conditions (paralysis, convulsions, which persist for months or even during life). Catarrhal pneumonia often generates chronic degenerative processes the clinical tableau of which is that of pulmonary phthisis.

The average mortality is from 50 to 60 per cent. As serious prognostic elements let us mention : young age, weakness, anemia, a simultaneous extension of the process to different organs, convulsions and paralysis, lobular pneumonia, colliquative diarrhea, intense and stationary hyperthermia, rapid emaciation, exhaustion, a fetid exhalation, finally a lowering of the temperature. Death is generally produced by cerebral paralysis (the latter may determine it within a few days), by pulmonary cedema, septicemia, or exhaustion. Among favorable prognostic circumstances we must particularly mention : old age, mildness of the process and its localization to circumscribed regions or its moderate extension. According to a few authors, cutaneous exanthema would constitute an unfavorable prognostic symptom. The facts which we have been able to observe do not at all agree with that assertion.

Differential diagnosis. We must especially differentiate this disease from simple mucous and primary catarrhs. This distinction is not always possible, for the catarrhal form of this process may become localized in only one organ. In general, however, we have as a guide the multiplicity of catarrhal determinations, a simultaneous invasion of several organs, young age of affected animals, the intensity of the fever, the mode of evolution of the disease, and especially the pustular eruption, which is here of great importance. In some subjects we notice phenomena of cerebral excitement, which often make owners fear the existence of hydrophobia. But the aggressive tendencies so characteristic of rabies are wanting, and the course of the disease is soon significant. When the exanthema extends to large surfaces it may offer a certain resemblance

to mange; pruritus is moderate or absent; the eruption rapidly extends to the whole surface of the body; finally, other symptoms proper to canine distemper exist which should sufficiently enlighten the practitioner. There are cases where mange and a specific exanthema exist simultaneously; here the diagnosis requires a careful examination. The acuity of the epileptiform complications which occur during the course of the disease enables us to distinguish them from true epilepsy.

Pathological anatomy. The respiratory apparatus shows the alterations of rhinitis, laryngitis, bronchitis, and catarrhal pneumonia. The nasal mucous membrane, which is sometimes pale, at other times dark red, is tumefied and covered with a purulent, thick, greenish or brownish-gray exudate mixed with blood-clots; this exudate is found accumulated between the lamella of the turbinated bones and in the frontal sinuses; in some cases it is covered with hemorrhagic ulcerations. The laryngeal and bronchial mucous membranes, which are red, tumefied, and ecchymosed, are coated with a muco-purulent layer; sometimes we notice in it catarrhal ulcerations. In the large bronchi we often find but slight alterations compared with the intensity of the symptoms observed during life; hyperemia and tumefaction of the mucous membrane of these ducts may disappear rapidly after death. The bronchioles are ordinarily filled with a purulent, dirty gray or bloody matter. The surface of the lungs is studded with reddish islands; certain regions are deficient in breathing surface, or are even entirely collapsed, others are distended with air. The centres of catarrhal pneumonia are characterized by their firm consistency, by the absence of air in their mass, by their protrusion on the surface of the organ, and by their reddish-brown or grayish-red coloration; their section is smooth or finely granular, on compression a turbid liquid exudes, which resembles chocolate when the lesions are of recent origin; it is reddish-gray or pale green when they are of long standing. In very young animals we usually find a vast pulmonary area infiltrated with a soft fibrinous exudate which easily liquefies (croupous lobar hepatization). Sometimes the inflamed islands are overrun with numerous small miliary centres; in other cases we observe therein a diffuse purulent infiltration. On the surface of the hepatized regions the pulmonary pleura is usually phlegmosed, the bronchial ganglions are tumefied and infiltrated with serum or pus.

The gastro-intestinal mucous membrane, especially that of the

intestinal grêle, is red, tumefied, studded with hemorrhages, and covered with viscous mucus; in some cases, however, it is pale, tumefied, friable, and coated with a layer of creamy appearance. The intestinal contents are often bloody. The mesenteric ganglions are tumefied.

The encephalon mostly shows alterations of cerebral œdema: anemia, soft consistency of the nervous substance, flattening of the convolutions, dampness and dull aspect of the section; there is a serous exudate in the lateral ventricles and in the subarachnoid spaces. Simple venous hyperemia of the brain is more rare; it is marked by distention of the sinuses, vascular plexus and bloodvessels of the pia mater, by the existence in the sections of numerous minute drops of blood, which may be removed by wiping. Kolesnikoff, who has made a histological study of these lesions, has observed a leucocytic infiltration of the cerebral substance, and especially of the vascular walls. Krajewski has mentioned, besides a dilatation of the cerebral bloodvessels, the accumulation in the perivascular spaces of lymphoid elements, which are sometimes found in the track of the brain as far as the protoplasma of the ganglionic cells. Alterations of the spinal cord are less prominent; ordinarily there is but little anemia and a slight œdema of the lumbar region. In cases of acute paralysis Mazulewitsch has noticed degenerative alterations of the vascular walls as well as the existence of an albuminous exudate along the bloodvessels and in the interstitial connective tissue of the gray substance; in the chronic form this author has found a circumscribed interstitial myelitis with partial atrophy of the nervous substance. According to Nadden, there exist in the latter masses of migrated red corpuscles.

We must also mention a quantitative anemia accompanied by a certain degree of hydremia, turbid tumefaction, and fatty degeneration of hepatic and renal cells, a grayish-yellow coloration of the myocardium (due to segmentation and fatty degeneration of the muscular fibres), œdematous tumefaction of the lymphatic ganglions, a muddy, viscous consistency of the blood (septicemia complication); finally, eschars upon the projecting regions (elbows, thighs, etc.), effect of prolonged decubitus.

Treatment. This is principally symptomatic. We have, however, certain medicinal agents which are capable of destroying the contagious germs on the spot. Our researches have established

that calomel enables us to combat the disease effectively in cases where it is localized in the digestive apparatus or when the infectious elements have penetrated by that duct. In the gastric form and in the beginning calomel, administered twice or three times daily in a dose of 0.05 gramme, has given us remarkable results. It acts by disinfecting the gastro-intestinal mucous membranes. We may also practise disinfection of the respiratory apparatus by means of cresol inhalations (1 per cent.); they are of advantage in the bronchial form; we have also obtained excellent results from it in pulmonary manifestations. But we are mainly restricted to symptomatic medication.

1. Ocular complications should be treated according to indications which are given in surgical works. Numerous observations have shown us that cresol water ($\frac{1}{2}$ to 1 per cent.) is the best agent to combat ulcerations of the cornea and purulent conjunctivitis. In this latter we also use sulphate of zinc ($\frac{1}{2}$ to 1 per cent.), and in obstinate cases nitrate of silver ($\frac{1}{2}$ to 2 per cent.), taking care to render harmless the excess of this medicament by washing with a solution of chloride of sodium. Ulcerations may also be combated by a 2 per cent. boric acid solution. In cases of parenchymatous keratitis we make use of solutions of atropine (sulphate of atropine, 0.1 gramme; water, 200 grammes). For conjunctival pruritus we use solutions of cocaine (2 to 5 per cent.); we thus avoid ulcerations of purely traumatic origin—due to scratching—and the cocaine also exerts upon the mucous membrane an antisecretory action (hydrochlorate of cocaine, 0.5 gramme; distilled water, 10 grammes—5 drops in eye every three hours). In cases of blennorrhoea we may cauterize the inflamed mucous membrane with nitrate of silver or sulphate of copper. If the trouble is obstinate, we must use the thermo-cautery after having previously anesthetized the eye with cocaine. Long-standing opacity of the cornea yields sometimes to insufflations of calomel or to red precipitate ointment (1 p. 15–25). Staphyloma and entropion require surgical treatment.

2. When the disease is localized in the digestive apparatus emetics are often of advantage in the beginning (hydrochlorate of apomorphine, 0.005 gramme to 0.01 gramme; water, 1 gramme to 5 grammes—in subcutaneous injection). As a stomachic we usually administer hydrochloric acid (per drops) alone or associated with bitters (hydrochloric acid, 5 grammes; tincture of gentian, 20

grammes; distilled water, 150 grammes—half a tablespoonful or a teaspoonful three times a day). We may also resort to rhubarb (tincture of rhubarb in a water or wine solution, to be given per teaspoonful); to decoctions of quinquina (10 to 150 grammes—to be given per tablespoonful), either pure or with the addition of a few drops of hydrochloric acid, with subnitrate of bismuth (0.1 gramme to 0.5 gramme), with meat extract (2 grammes to 2.5 grammes), with wine (per teaspoonful or tablespoonful), etc. Obstinate vomiting may be arrested by opium, chopped ice; and diarrhea, with tincture of opium (plain tincture of opium, 0.5 gramme to 2 grammes), opium powder (0.1 gramme to 0.3 gramme), tannin (0.2 gramme to 0.5 gramme), nitrate of silver (0.02 to 0.05 gramme), dissolved in distilled water or given in pills. For bloody diarrhea we administer opium associated with gummy preparations (plain tincture of opium, 10 grammes; gumarabic, 20 grammes; distilled water, 250 grammes—to be administered three times daily per teaspoonful or tablespoonful).

3. The treatment of localized areas in the respiratory apparatus is similar to that of laryngitis, bronchitis, and catarrhal pneumonia. When the cough is frequent we must resort to morphine preparations (hydrochlorate of morphine, 0.1 gramme; bitter-almond water, 10 grammes; to be given three times daily per teaspoonful or tablespoonful); we must besides apply moist and hot compresses upon the larynx and chest. Bronchial diseases require expectorants: hydrochlorate of apomorphine (0.01 gramme *per die*, *per os*), tartar-emetic and golden sulphuret of antimony (0.02 gramme to 0.05 gramme; this drug must be reserved for robust animals); hydrochlorate of ammonia (0.1 gramme to 0.5 gramme), tartar-emetic (wine of antimony, per drops), *Polygala senega* root (decoction of 10:150; to be administered per teaspoonful or tablespoonful), ipecac (0.02 gramme to 2.05 grammes).

4. Spasms originating from the central nervous system are combated by antispasmodics, with bromide of sodium (10 grammes; water, 250 grammes; three times daily per tablespoonful or teaspoonful), urethane (2–20 grammes), hypnone (0.25 gramme to 2 grammes), sulphonal (1–4 grammes), hydrate of chloral (0.05 gramme to 5 grammes in a mucilaginous solution), morphine (subcutaneous injections of 0.02 gramme to 0.1 gramme). Paralytic conditions and weakness are treated with stimulants: coffee (infusion of 5–10 grammes to 100 grammes of water), broth, extract of

meat, wine, camphor; hypodermatic injections of camphorated alcohol or oil (5 to 10 grammes), ether (1 gramme per hour), caffeine (0.5 gramme to 2 grammes), hyoscyamine (0.005 gramme to 0.02 gramme), atropine (0.01 gramme to 0.05 gramme), veratrine (0.001 gramme to 0.005 gramme), strychnine (0.001 gramme to 0.003 gramme), electric current (Spamer's apparatus).

5. Special treatment must be instituted for hyperthermia when this condition is pronounced and portends danger for the important organs, especially for the heart. The fever at the beginning and slightly elevated temperature (39°–40° C.) do not require any particular medication. Among the antifebriles we may use in preference antipyrine, antifebrine, and phenacetine; the action of these agents is certain, and is produced in a relatively short time. They are administered in a watery solution, in a dose of 25 centigrammes to 1 gramme per hour, till the temperature has returned to normal. Quinine is less safe in its effects. Hydrocyanic acid, in the form of bitter almonds (1 gramme to 2 grammes *per dosi*, every two to three hours), is also an anti-thermic upon which we may rely.

6. Cutaneous alterations require very little care; the moist regions may be dried with powders (oxide of zinc, 1 gramme; starch, 10 grammes); glycerin is of advantage in softening the crusts produced by the pustules.

From a dietetic standpoint it is proper to give alible and easily-digestible food: raw chopped meat, milk, broth, are very advisable. The prophylaxis requires that the sick shall be separated from the healthy animals.

Disease of cats is identical with the affection we have just studied; Krajewski has demonstrated it experimentally by inoculation from one species to the other. In the cat the disease is also observed in an epizootic form. The symptoms are nearly the same as in the dog, except nervous symptoms are extremely rare—almost unknown. Vomiting, inappetence, depression, purulent conjunctival discharge, sneezing, cough, dyspnoea, wheezing respiration, diarrhea, complaints, great weakness, anemia, comatose phenomena, and fall of temperature are its principal manifestations. Exanthema seems to be wanting. The alterations recognized at the autopsy are similar to those of the disease of the dog; in the lungs we have found marked alterations, especially those of croupous lobar pneumonia. The treatment is the same as in the dog. We must,

however, proscribe inhalations of phenic acid, by reason of the toxic effect of this agent on the cat. The doses of medicaments should be about one-fifth of those indicated for the dog.

MALIGNANT CATARRHAL FEVER OF THE OX.

Headache: Gangrenous Coryza.

Etiology. Malignant catarrhal fever is an infectious disease which is exclusively seen in the ox, and the agent of which is unknown. Its contagiousness is very slight; direct contamination is very rare; all attempts at inoculation have remained without result. It is very probably produced by a "stable miasma," which is especially developed in defective and badly-kept stables. Cases have been reported establishing its transmission by certain intermediaries: by the sheep (Möbius), by cattle dealers (personal observations). As a rule, malignant catarrhal fever is sporadic, but its extension to all bovines of one stable, or even of one locality, is not rare; it is stationary in some countries; it may also exist for several years in succession on the same premises. A large number of cases of this sort are mentioned in the publications. Sump has observed it for twenty-five successive years on a large farm; out of two hundred and twenty-five cows which were affected, only three escaped death.¹

It mostly affects young animals which are well fed, though it is also observed in adult subjects. Spring seems to be the most favorable season for its development. According to Frank, it is particularly frequent in localities with light soil which is moderately damp, and does not exist in regions where the lower layers of ground are very dry or very damp. But nothing very precise is known about the influence which is exerted by cosmo-telluric conditions; we may find the trouble at all altitudes—upon hills, on the plains, and in valleys—no matter what the condition of the lower ground of these localities may be. Cold acts only as a predisposing cause. The duration of the incubation is from three to four weeks (Bugnion, Frank).

According to the localization and intensity of the morbid state we have for a long time recognized the *nasal*, *abdominal*, *exanthematous*, *benign* and *malignant* forms. The multiplicity of forms

¹ Sump: Preuss. Mittheil., 1857.

which it may assume has given rise to very divergent opinions on this subject.

Only recently Brusasco has made attempts at transmission, the results of which were negative. This experimenter used on the muzzle and pituitary mucous membrane blood, nasal mucus, and exudates coming from the sinus; he has also caused the ingestion of these products without obtaining any positive result. Cohabitation has not been more efficient.

Zündel and other authors have affirmed the identity of malignant catarrhal fever with tuberculous basillary meningitis. This opinion is erroneous, for the anatomical alterations and symptoms of these diseases differ considerably. Besides, tuberculous basillary meningitis is almost exclusively observed in poor animals (because they are tuberculous), while catarrhal fever is usually seen in young and strong animals.

Symptoms. Catarrhal fever is marked by a symptomatic *ensemble* which is very variable. It is a constitutional disease, in which the ocular, respiratory, digestive, and genito-urinary mucous membranes are the seat of several inflammatory processes (catarrhal, croupous, diphtheric). It is accompanied by serious phenomena, and especially by cerebral complications. In some instances only one organ is affected; in others different organs may be attacked simultaneously.

1. *Beginning period.* The disease is generally announced by chills and intense fever; from the first or second day the temperature oscillates between 40° and 42° C. The animals are depressed; the head is supported on the manger or it is hanging and inert; the base of the horns, the cranium, and sinuses are hot; there are chills and generalized muscular trembling; the hair is dull and bristly; the back is often arched. Sometimes within a few days emaciation makes rapid progress.

2. *Ocular troubles.* The first is abundant weeping from the eyes; soon the conjunctiva assumes an intense red color, the eyelids become tumefied, and photophobia produces occlusion of the organ; tumefaction may determine true ectropion. The conjunctivitis is complicated later by a diffuse keratitis; the cornea becomes turbid in its periphery; it is at first of a suspicious aspect, later it becomes milky white. Inflammation of the iris, which is quite frequent, is accompanied by an exudation into the anterior chamber of the eye (exudative iritis); this membrane at times becomes joined to the

crystalline capsule (adhesive iritis). In some instances these symptoms disappear gradually; in other cases the cornea becomes perforated and the iris forms a prominent hernia; finally, blindness may occur through atrophy of the bulb. If considered in its *ensemble* the process shows quite a marked analogy to the periodical ophthalmia of the horse.

3. *Troubles of the respiratory apparatus.* On the respiratory mucous membrane there is developed a phlegmasia at first catarrhal, and which later becomes croupous or diphtheric (necrosis). The nasal mucous membrane, which is intensely red or bluish-red, is covered by croupous or diphtheric products which leave after them wounds of an ulcerative character. At the same time appears a discharge which at first is sero-mucous and later purulent, clotty, striated with blood, of bad aspect, and fetid. Respiration is difficult, rattling and snoring; auscultation shows the presence of mucous râles. Dyspnœa becomes accentuated; we may observe attacks of true suffocation when the larynx and nasal cavities are obstructed by false membranes. Sometimes croupous masses are ejected outward.

When the inflammatory process reaches the frontal sinuses in the cavity of the horn it affects the matrix of this organ, which fills the office of periosteum to the bony core. The horns are very sensitive to the touch; they may easily be pulled out or even become loose and fall spontaneously.

4. *Troubles of the digestive apparatus.* In serious cases the appetite is lessened from the beginning, but in the benign form, and when the disease does not involve the digestive organs, it may persist for quite a long time. During the first days the buccal mucous membrane is red, dry, and hot; later stomatitis determines ptialism. Saliva drops on the soil in long threads. The mucous membrane is overrun with hemorrhages, and presents erosions in the palate, upon the bars (superior maxillary), and on the cheeks; we observe also a grayish-yellow or whitish-yellow diphtheric exudate, which is disposed in islands, the removal of which exposes ulcerated surfaces. The necrotic inflammation may spread to the skin of the muzzle or nose; the whole face is then considerably tumefied. The buccal cavity exhales a fetid odor. We observe also an alternation of constipation and diarrhœa with the ordinary symptoms of colics. The excrements are very thin and fetid; they often contain blood and croupous neoformations; sometimes the patients eject membranous

products which are from two to three metres long and detached from the intestinal mucous membrane (Frank). There is tenesmus; the rectal mucous membrane is much tumefied.

The lacteal secretion is usually entirely suppressed within a few days.

5. *Troubles of the genito-urinary apparatus* are those of nephritis and cystitis. The patients make convulsive efforts in order to micturate, which are very painful. The urine frequently contains blood, albumin, and characteristic products of nephritis (urinary cylinders, renal epithelium, white corpuscles). According to Frank, its reaction is often acid. The vaginal mucous membrane, which is congested and tumefied, is often covered with diphtheric exudates or strewn with ulcerations. The vulva discharges a mucons grayish or bloody liquid. Most females which are seriously affected abort.

6. *Nervous symptoms.* We mostly observe phenomena of cerebral hyperemia and encephalitis. In many cases they dominate the scene. The animals are excited and restless; they climb into the manger, bellow, lean or throw themselves sideways; the eye is haggard; we observe at times rabiform and spasmodic attacks which are variable in their intensity; convulsions, epileptiform spasms, twitching of the eye in its orbit, and trismus. These convulsions are followed by paralysis and coma, the temperature falls, and death is near at hand.

7. In some cases the skin shows a pustular eruption; the hairs fall out, an abundant epidermic desquamation is produced on the udder, on the inner fascia of the joints, between the digits, on the neck, back, etc. We have several times observed the falling out of the hoofs.

Pathological anatomy. Like the symptoms, the anatomical alterations vary with the localization, the age, and the degree of intensity of the morbid process. The lesions which are noticed in slaughtered animals differ considerably from those shown at the autopsy of subjects which die; in the former we especially find catarrhal and croupous alterations; in others we are more likely to meet with true diphtheric processes. Let us examine the most important.

The nasal mucous membrane is brownish or bluish-red, tumefied, ulcerated, and studded with hemorrhages or covered with yellowish-white croupous membranes. The turbinated bones and ethmoidal

cells are sometimes affected with necrosis. The mucous membrane of the frontal sinuses, which is thickened, is coated with a purulent exudate; the matrix of the horns is inflamed and their cavity is filled with pus. The laryngeal and bronchial mucous membranes show lesions of catarrhal, croupous, or hemorrhagic phlegmasia. The croupous exudate quite often extends as far as the finest bronchi. When the disease has ended in death we frequently observe in the lung either interstitial emphysema or diffuse œdema.

The buccal mucous membrane is bluish-red and tumefied in the neighborhood of the teeth. Gillmayr has found therein vesicles which are rapidly transformed into superficial erosions. These latter are quite common; they may reach and even exceed the dimensions of a ten cent piece. In serious cases the palate and its velum are covered with yellowish-white membranous islands; sometimes they exist also in the pharyngeal mucous membrane and on that of the œsophagus at the origin of this canal. In the pharynx we find alterations similar to those of the mouth. The gastrointestinal mucous membrane is of a diffuse or marbled red color, tumefied and strewn with diphtheric exudates and ulcerations which leave indelible cicatrices. Peyser's patches and the solitary follicles are generally ulcerated, an alteration which gives to the disease a certain resemblance to bovine plague. The encephalic meninges are hyperemic and infiltrated; the brain and its plexus are œdematous and show hemorrhagic centres. The spinal cord has undergone similar alterations.

The kidneys are inflamed and undergoing fatty degeneration. In the pelvis we find, at times, a croupous exudate which may continue along the ureters as far as the bladder. The mucous membrane of this viscus is inflamed to a variable degree, is quite often covered with croupous membranes, and is marked by hemorrhagic centres.

As general alterations we observe: hemorrhages in the different organs, but especially in the heart, the serous membranes, and more particularly in the mesentery and the epiploon; fatty degeneration and decoloration of the muscles, rapid decomposition of the cadaver, fluidity and viscosity of the blood, accumulation in the splanchnic cavities of a bloody liquid and a slight inflammation of the pleura.

Course and prognosis. According to the cases the course is hyperacute, acute, subacute, or chronic. At times death is produced within three to five days; at other times a cure is obtained after many weeks. The average duration of the disease is about one

month. Relapses are frequent, and there is no way of foreseeing them; sometimes a first recrudescence takes place from the second to the third week. Frequently, in the beginning, in cases when we think that we observe symptoms of improvement the disease becomes suddenly aggravated. Convalescence is always prolonged. In benign cases a complete cure is often obtained in a month; in the serious form, when the disease does not terminate rapidly by death, its duration is two to three months.

At all periods the prognosis is doubtful, and should be expressed with great reserve. The mortality varies from 50 to 90 per cent. In 76 patients observed by Bugnion, 7 recovered; the others were slaughtered the third or fourth day. Concerning animals which were subjected to treatment, Frank estimates that the successes do not exceed 6 per cent.

The unfavorable prognostic symptoms are the extension of the process to all the organs or to a certain number of them, the intensity of the inflammation and necrosis of the affected mucous membranes, serious cerebral, pulmonary, or intestinal complications. As chronic morbid conditions generated by the disease, let us point out the alterations of the eyes above described, and catarrh of the sinuses.

Differential diagnosis. It is especially important to distinguish catarrhal fever from *bovine plague*, which is far from being always easy. Differentiation is based upon the following facts: 1. Catarrhal fever is but slightly contagious; bovine plague, on the contrary, is extremely contagious. 2. In the former the ocular and respiratory troubles (contraction bruit, discharges, etc.) ordinarily predominate; in bovine plague gastric complications dominate the scene; to these peculiarities are added the different lesions found at the autopsy. 3. In malignant catarrhal fever the course is generally less acute than in bovine plague. But when the former simultaneously affects several animals in the same stable, and when the process becomes especially localized in the digestive apparatus and the vaginal mucous membrane, the symptoms may mislead the practitioner. In this case the diagnosis is explained by the history and by circumstances which should make us admit or reject the possibility of the invasion of bovine plague. We have sometimes resorted to inoculation in the ox; the negative result indicated that catarrhal fever was present.

It may also be confounded with dysentery, periodical ophthalmia,

and meningitis (Zundel, we have said above, admitted the identity of catarrhal fever with tuberculous basilar meningitis).

Treatment. As prophylactic measures, Frank advises disinfection and drainage of the lower ground of infected stables; he also recommends the interposition of an impermeable layer between the earth and the pavement. The curative treatment formerly used comprised the following remedies: bleeding, lotions and fumigations of vinegar to the skin, amputation of horns, trephining, tartar emetic and camphor administered internally. More recently phenic acid, administered in a dose of 5 to 10 grammes per day, and used in fumigations or in frictions, has been recommended. In cases of serious cerebral complications we make refrigerant applications to the head (ice, cold compresses and douches). We may also institute a symptomatic treatment (antipyretics, alkalies, expectorants, emollients).

Malignant catarrhal fever has nothing in common with *diphtheria of man*, contrary to what has sometimes been advanced. (See Diphtheric Diseases of Domestic Animals.) Human diphtheria is an infectious disease *sui generis*, the etiology, contagiousness, symptoms, and lesions of which differ radically from the disease we have just described. Diphtheric or necrotic inflammations of the mucous membranes may appear during the course of very dissimilar diseases.

INFECTIOUS ABORTION OF THE COW.

Epizootic Abortion.

Etiology. Abortion in bovine animals is observed in an epizootic state in certain regions and stables. It has been known to exist permanently on some farms, and to cause for many years serious pecuniary loss. This enzootic or epizootic character of an accident which is usually produced by trivial causes, such as contusions, acute febrile diseases, cold, poisoning, ingestion of tainted or mouldy food, and unwholesome drinks indicates that it is sometimes of an infectious nature. As long as its pathologic agent is not known, epizootic abortion must be classified in the group of specific diseases.

The infection seems to be produced by a "stable miasma." It has been demonstrated that the virulent agent exists in the dis-

charge of the genital canals and in the foetal fluid; by the intermediation of these liquids healthy animals may be affected. By introducing into the vagina of healthy cows the vaginal mucus of a cow which had just aborted, Bräuer produced abortion in eleven cases; the accident occurred from nine to twenty-one days after inoculation. By proceeding in the same manner Lehnert has caused abortion twice within twelve to twenty days. Trinchera has produced a purulent vaginal catarrh and abortion in from nine to thirteen days in healthy cows which he had inoculated with the purulent vaginal exudate of a patient; the inoculation matter obtained by scraping the surface of the chorion expelled by a cow which had aborted caused a like result. Moreover, experience has sufficiently demonstrated that the disease is eminently contagious, and that it may be transmitted directly or by certain intermediaries: through the lochia, litter, by persons who are charged with the care of the patients, by the veterinarian (after extraction of the placenta from an animal which has aborted), and even by breeding males.

The transmission of the disease from a cow which has aborted to its immediate neighbors is the rule, and it is much favored by the existence, behind the animals, of a trench where the lochia and excrementitious matters accumulate.

The causes to which the disease was formerly ascribed—tainted food, rainy years, bad quality of food, permanent stabling, close breeding, etc.—are but predisposing conditions. By weakening the organism they facilitate the introduction and pullulation of the infectious matter. According to Strebel, in the district of Freiburg (Switzerland), during the wet years of 1878 and 1879, 20 to 60 per cent. of impregnated cows aborted. Epizootic abortion may establish itself in the best kept stables—a fact which proves that uncleanliness plays but a secondary etiological rôle. A decomposition of the after-birth alone could not determine it, as the relative rarity of abortion, in comparison with the frequency of non-delivery and decomposition of the envelopes, is a fact in opposition to this theory. Nothing positive is as yet known as to its pathology. It is very probable that the infectious agents penetrate into the womb through the vagina and os, like the septic elements in parturient fever; their pullulation in the foetal envelopes determines sufficient alterations to lead to abortion. The process is, no doubt, propagated from the covering to the foetus, as the death of

the latter seems to demonstrate *ante-partum* in most cases of epizootic abortion. At the present time it is not known whether the virus may penetrate into the blood through the respiratory or intestinal tracts. Biot considers as cause of abortion an infectious inflammation of the serous coating of the uterus. Nocard claims that the specific agent multiplies between the uterine mucous membrane and the chorion, that it does not exercise any noxious influence upon the former, but that it attacks the envelopes after each new conception; it would thus determine repeated abortions, and lead to sterility by communicating to the uterine secretion an acid reaction which is fatal to the spermatozooids.¹

¹ Nocard has specially studied epizootic abortion from an anatomo-pathological and bacteriological standpoint. The autopsy of diseased animals does not reveal any alteration of the thoracic or abdominal viscera, with the exception of the uterus. The peritoneal serous membrane is normal: incision of the uterus reveals the presence, between the mucous membrane and the chorion, of a fibrinous, muco-purulent matter which is more or less abundant and often acid, in which the microscope shows epithelial cells, leucocytes, a large number of isolated micrococci which are generated or associated in short chains of three, four, or five sections, and a few short, thick bacilli which are isolated or associated in pairs; in the cotyledonous liquid the bacilli are predominant; in the product obtained by scraping the uterine mucous membrane both micro-organisms exist in almost equal number. The amniotic liquid also contains them. In the aborted young the intestinal mucous membrane is the seat of abundant epithelial desquamation; its tissue seems infiltrated with various microbes, which exist in abundance in the contents of the intestine. These micro-organisms which are contained in the digestive tube give an explanation of the diarrhea by which they are affected in the two or three days following their expulsion: these calves have been aborted in an advanced period of gestation. In the aborted young, which bellow continually during the days preceding death, the medulla contains micrococci which are identical with those contained in the amniotic fluid.

From his researches the author concludes as follows:

"1. In aborted cows, even in primipara, there exist in the uterine cavity, between the mucous and the fetal membranes, especially in the cotyledonous crypts, several micro-organisms which cannot be found in the fecundated cows, even in such as have already calved, when they come from a locality where this abortion does not exist.

"2. These micro-organisms do not seem to exercise any noxious action upon the uterine mucous membrane of the mother either during the period of gestation, which must be interrupted abruptly, or after abortion.

"3. Repeated abortion in the same subject can easily be explained, if we admit the pathologic influence of a microbe, by the persistency of the latter in the uterine cavity till the time when it is able to exercise this action on a new fetus or on its envelopes.

"4. Also, cases of sterility which are consecutive to abortion can be explained by the acid reaction of the uterine mucus in which the microbes thrive, the spermatozoa not being able to preserve their vital properties except in alkaline elements.

"5. Epizootic abortion seems, indeed, to be a microbic disease of the fetus and its envelopes, but one which does not affect the mother."

He indicates the prophylactic treatment thus:

Epizootic abortion is also observed in the sheep and goat,¹ and more rarely in the mare.

Symptoms. In the cow abortion generally takes place from the third to the seventh month of gestation; in the mare, from the fourth to the ninth month. The prodromes are: a redness of the vaginal mucous membrane, on which we frequently observe eruptions in the form of pimples, about the size of a millet-seed, discharge from the vulva of a reddish liquid, and a lessening of the lacteal secretion, which acquires the consistency of the colostrum. Three

"1. Every week the floor of the stable should be scraped, thoroughly cleansed, and sprinkled with a solution of sulphate of copper of the strength of 40 grammes per litre.

"2. Every week we should inject with considerable force, by means of a horse-syringe, into the vagina of cows with calf a syringe-ful of the following tepid liquid:

" R.—Distilled water	20 litres.
Glycerin	} 5ā 100 grammes.
Alcohol at 36°	
Bichloride of mercury	10 "

"Dissolve the bichloride of mercury in the alcohol and glycerin; mix with the water and shake thoroughly. This solution should be kept in a wooden vessel, barrel, bucket, or pail, and must be kept out of the reach of children or animals.

"3. Every morning, at the time of grooming, we must carefully wash the vulva, anus, and the inner fascia of the tails of all the cows with calf, with a sponge that has been saturated with the same tepid solution.

"4. Finally, in case a cow should abort, it is necessary: *a*, to deliver it immediately with the hand; *b*, to destroy by fire or boiling water the fœtus and placenta; *c*, to irrigate the uterine cavity by means of a long rubber tube, conducted by the hand to the fundus of the organ, using 8 to 10 litres of the tepid solution indicated above." (See *Receuil Vét.*, 1886.)

Galtier, de Poncius, and Ory have studied epizootic abortion on an extensive farm where it has existed for more than twenty years. According to these authors, epizootic or infectious abortion is the consequence of a microbic disease, of a general infection in the mother, which communicates to the fœtus the disease by which it is affected. This disease confines itself almost exclusively to bovine animals, but it is transmissible to the various species of domestic animals, and presents a few characters which are common to pneumo-enteritis of the pig (pneumo-enteritis of Gentilly) and that of the sheep (pneumo-enteritis of the Alps). (See *Journ. de Lyon*, 1890.)—N. D. T.

¹ Labat has reported the history of an enzooty of infectious abortion which existed in a flock of sheep in the Department of Aude. This flock, which consisted of 86 sheep, four, five, and six years old, were stabled in a narrow, low, and badly-kept sheepfold. Within three weeks 15 of these animals with lambs nearly three months aborted without any known cause. The author, who was consulted, prescribed a treatment comprising as principal indication: vacation of the sheepfold, separation of the healthy females from those which had aborted, disinfection and careful cleaning of the premises, destruction of the aborted young and placenta, and every morning a sponging of the anus, vulva, perineum, and tail with a solution of 1:2000 corrosive sublimate (sublimate, 1 gramme; alcohol, 100 grammes; boiled water, 2 litres). The epidemic disappeared within a week. (See *Revue Vét.*, 1889.)—N. D. T.

days after the appearance of the discharge the abortion takes place and gives rise only to insignificant general symptoms ; the foetus is usually dead. After the accident there persists quite frequently a vaginal hypersecretion. Sometimes the mother suffers for a long time ; she may become sterile.

Treatment. The *prophylaxis* is important. Once started, it is impossible to prevent the abortion ; opium and other medicaments are powerless. We must, first of all, isolate the sick animals ; it is advantageous to put the healthy cows at pasture. It is also indicated to destroy the after-birth and the foetus, and to disinfect the stable and patients. For the latter we must have recourse to spongings or to vaginal injections with phenicated or cresol water (0.5 gramme to 2 grammes per 100). Cows with calf should be treated in the same way. We may also try to increase the resistance of the animals by means of food rich in nitrogen, by the administration of preparations of iron, and by exercise. But no matter what is done, very frequently abortion continues to occur. As prophylactic means, Bräuer has tried subcutaneous injections of a 2 per cent. solution of phenicated water. From the fifth to the eighth month of gestation he injected every fortnight the contents of two to three Pravaz syringes of this agent under the skin of the flank ; he is said to have obtained satisfactory results in a certain number of cases.

Experiments made more recently with phenic acid have given variable results ; they were of advantage in some cases, and null in others (Schleg). Theoretically, phenic acid seems to be inefficient ; in the organism it is rapidly transformed into sulpho-phenic acid, which is without effect.

Females which have aborted should only be used for breeding purposes after the disappearance of the vaginal discharge. Some authors have observed that a change of bull may have a favorable influence ; this has been explained by the better constitutional condition of the progenitor ; it depends perhaps upon the fact that the male sometimes becomes an agent of transmission of the disease.

DYSENTERY OF YOUNG ANIMALS (DYSENTERIA NEONATORUM).

GENERAL CONSIDERATIONS. Dysentery of the newly-born is the least known of all infectious diseases of animals. In ancient

works the name of dysentery is given to a series of pathological conditions of very different nature: intense intestinal catarrh with diarrhea, "dysenteroid," enzootic and toxic enteritis (in the ox), bovine plague. Our knowledge of the nature of true dysentery in adult animals is very incomplete. If in the ox there have been published the several facts which appear to be related to this trouble, none have been mentioned in the horse, the sheep, and the dog. Some cases of dysentery have been described in the dog, but they are not of an authentic character. The dysentery of chickens seems to be dependent upon cholera or upon diphtheric processes (see Typhoid of Birds).

Dysentery of sucklings (calf, lambs, foals) is an infectious disease which presents a certain analogy to dysentery of man; up to the present time, however, nothing establishes the identity or the degree of affinity of these diseases.

Dysentery of man consists essentially of a croupous or diphtheric infections inflammation of the large intestine, with purulent or hemorrhagic infiltration of the mucous membrane and subjacent connective tissue. Its specific agent is as yet unknown. It does not appear to be transmitted by direct contagion. Contamination takes place through the excrements of affected animals. Dysentery is sporadic or enzootic; while in our climate it has generally a benign course, it is very serious in tropical regions. Its principal symptoms are: colics, tenesmus, vomiting, a mucous, purulent, or hemorrhagic diarrhea, which becomes more and more abundant, also dysuria and constitutional symptoms of variable gravity. Sometimes we observe complications: hepatic abscesses, articular diseases, inflammation of the serous membranes, perforative peritonitis, etc. Administration of purgatives (calomel, castor oil) and stimulants constitutes the principal indication.

Etiology. Dysentery of the newly born is observed in the calf (dysentery of calves, white dysentery), lamb, foal, dog, and cat. This affection and pyemic polyarthritis are the most fatal diseases of young age. According to Röhl, it is so common in certain districts of Austria (Salzbourg, Saint Jean, etc.) that in one region, in a total of 3318 calves, 1196 have been affected by it, of which 1152 (97 per cent.) died. In 1884, in the same districts, its mortality was 55 per cent. In general it is developed from the first to the third day following birth; after the fourth day it is much less frequent; young animals are often affected by it before having

sucked; milk, therefore, has nothing to do with the development of the disease. Its exciting cause is evidently an infectious element yet unknown, which is contained in the excrements and is carried by them. It appears to be miasmatico-contagious, and seems to be transmissible from one animal species to another [from the calf to the lamb for instance (Kotelmann)]. Gutmann has not succeeded in communicating it by means of ingestion of excrements.

We have frequently remarked a coexistence in the same premises of epizootic abortion and dysentery of calves, and it has been concluded from this that causal relations exist between these two diseases. Franck admitted intra-uterine infection of the foetus by agents which would reach it by ascending the genital passages; he would thus explain the appearance of the disease in the calf during the first days of life. It is probable that intra-uterine infection occurs by the propagation to the digestive mucous membrane of young animals of an infectious product of the uterus and vagina.

Pathological anatomy. Anatomical alterations present nothing characteristic. The intestinal mucous membrane is covered with a muco-purulent exudate containing numerous bacteria; the epithelium is desquamated on certain regions; the chorion and Peyer's patches are tumefied and softened. In serious cases the intestinal contents are bloody. Ulcerative processes seem to be slight, no doubt by reason of the short duration of the disease. The mucous membrane of the abomasum is congested toward the upper edge of its folds, especially in its pyloric portion; it is ecchymosed and oedematous; it is also frequently found macerated. The abomasum generally contains caseous lumps of an acid reaction. We have often found therein traces of hemorrhages. A striking fact is the very marked anemia and parenchymatous alterations of the internal organs (liver, kidney) of the cadavers; in some cases we recognize also lesions of a lobular broncho-pneumonia which are produced by drinks that have miscarried.

Symptoms. The symptoms are almost similar in all species. The *calf* leaves off sucking; it shows symptoms of restlessness, bellows, and ejects, by violent efforts, very soft diarrhetic excrements which soon become liquid, whitish (mucous diarrhea), or mixed with clots of curdled milk (white dysentery); they are very often bloody; later, we notice continued and involuntary evacuations. The patients, which are exhausted, remain constantly recumbent; at intervals they are subject to convulsions; there is ptialism; the

expired air has a fetid odor. The animals often die within twenty-four hours, sometimes within three days. The mortality is very high (80 to 100 per cent.). In many cases all the calves of one stable perish. Those that survive remain for a long time weak and sickly.

The *lamb* leaves off sucking, it becomes depressed, very weak, and ejects mucous liquid excrements which have a fetid odor; it is frequently affected by tencismus. In the beginning the temperature rises to 41.5° C., but it afterward drops abruptly (Nikolski); the respiration is accelerated; saliva and mucus escape from the mouth. The disease appears usually during the three days which follow birth (an observation by Nikolski gives the following figures: 30 per cent. on the first day, 40 per cent. on the second, 25 per cent. on the third, and only 5 per cent. on the fourth and later). Its average duration is from one to three days; but in cases with a rapid course death may result within a few hours. In the *foal* the trouble appears during the first three days (Mazoux). The animals are depressed and restless; the excrements have a mucous or liquid appearance and are extremely fetid. The expired air and cutaneous exhalations often spread a disagreeable odor. The orbits become sunk; the patients are very weak, the thirst is intense, the belly shrunk; sometimes we observe a generalized or localized cutaneous eruption in the neighborhood of the anus.

Diagnosis. The diagnosis of dysentery is based on the existence of an intense diarrhea, which becomes rapidly fatal and which exists in an epizootic state. In sucklings it may be confounded with simple gastro-intestinal catarrh, which is due to a defective regimen or to alterations of the milk; but these catarrhs, which are generally mild, do not appear at a period so near birth.

Treatment.¹ The *prophylaxis* consists in separation of the healthy from the sick animals, and disinfection of the premises as well as the genital canals of the females before and after parturition. When dysentery of the calves exists in an enzootic state in a stable it is proper to place the cows with calves in an isolated and well-kept stable one or two months before parturition. Experience has taught that this means is much more efficient than all the medicaments which are employed in the treatment of the disease.

The first indication of treatment is the administration of a light

¹ See Gastro-intestinal Catarrh of Young Animals, vol. i., p. 94.

laxative (a few tablespoonfuls or teaspoonfuls of castor-oil for the calf and lamb; 0.1 to 0.2 gramme of calomel *pro dosi* for the colt).

Authors advise giving rhubarb root and opium, alone or together (rhubarb root in a dose of 2 to 4 grammes for the calf, of 0.5 to 1 gramme for the lamb; opium in a dose of 1 to 2 grammes for the calf, of 0.1 to 0.2 gramme for the lamb). We must recommend Hertwig's modified mixture: powdered rhubarb, 4 grammes; carbonate of magnesia, 1 gramme; powdered opium, 2 grammes—to be administered at one time to the calf in 100 grammes of infusion of chamomile or in 50 grammes of whiskey. We may replace the opium by tincture of opium (calf, 5 to 10 grammes; lamb, 1 to 2 grammes). Besides, we use tannin (calf, 1 to 2 grammes; lamb, 0.2 to 0.5 gramme), alone or mixed with an equal quantity of salicylic acid. In the calf we may use the following formula: tannic and salicylic acid, each 2 grammes—to be administered once or twice daily in an infusion of chamomile. Nitrate of silver has been commended (a tablespoonful of a $\frac{1}{2}$ per cent. watery solution every three hours for the calf), cresol (dose, one gramme in a watery solution three times daily for the calf), tar water ($\frac{1}{4}$ of a litre *pro dosi* for the calf), resorcin, etc. Mucilaginous preparations and protective substances (décoction of linseed, gum, marsh-mallow, mucilage, gelatin water, etc.), alone or associated with opium, may also be used.

Dysentery of Adult Animals.

Dysentery of adult animals has only been recognized in the ox. It affects in preference animals which are kept permanently stabled, and often disappears when they are turned out on pasture. It may be observed for several consecutive years in the same stable, but its intensity becomes lessened in the course of time. It is also found in a sporadic state. The causes to which it was formerly ascribed: cold, anomalies of regimen, excessive exertions, tainted food and drink, poor or rainy years, periods of war, etc., play the rôle of predisposing influences.

Pathological anatomy. The mucons membrane of the large intestine shows at several points the lesions of hyperemia. It is found tumefied, hemorrhagic, marked with swelling, deprived of its epithelium in certain regions, and covered in other places with a yellowish diphtheric exudate, which leaves behind ulcerations and cicatrices. The intestine contains fetid matters of bad aspect,

yellowish-gray or striated with blood. The microscope reveals numerous red corpuscles, leucocytes, and various microbes (Albrecht). The mucous membrane of the abomasum is tumefied and studded with hemorrhages; the lymphatic organs of the intestine, especially Peyer's patches, are the seat of great inflammatory infiltration or of an ulcerative process. In the chronic form the intestinal mucous membrane, which is thickened, shows at times a slaty-gray coloration; the muscular membrane is hypertrophied; on the surface of the ulcerations and cicatrices the serous membrane is altered by an adhesive inflammation.

Symptoms. The disease appears suddenly, and is marked by loss of appetite, a high fever (40° to 41° C.), weakness, colics, and a slight diarrhea. Later the excrements become liquid, mucous, frothy, fetid, and even bloody; they are often mixed with non-digested food, and with croupous membranes and desquamated epithelium. In the beginning we observe tenesmus and expulsive efforts, which are ineffectual, but which may determine rectal prolapsus; later, diarrheal matter runs continually from the gaping anus; the rectal mucous membrane is red and tumefied. Abdominal palpation is painful. Exploration of the right abdominal region permits us to recognize a pronounced fluctuation, which is due to the presence of an excessive quantity of liquid in the intestine (Albrecht). Weakness continues to increase, the temperature becomes lower, the patients show loss of flesh. In some instances death occurs in twenty-four or forty-eight hours, but more frequently from the second to the fourth day. Benign cases end in a cure within two or three weeks. Exceptionally the disease may be prolonged for months; this slow form appears to be related to intestinal ulcerations. The *prognosis* is serious and always uncertain. The mortality is about 50 per cent.

Differential diagnosis. In adult bovines we may confound dysentery with toxic, mycotic, enzootic enteritis (forest disease, browsing disease); often it is impossible to differentiate it from these diseases. Necropsic alterations frequently resemble those of bovine plague; but in dysentery the intestinal canal alone is affected, and we never observe any evidences of direct contamination.

Treatment. We must use mucilaginous preparations (decoction of linseed, marshmallow, etc.), opium (10 to 25 grammes), and astringents: sulphate of iron, gallie acid, nitrate of silver, sugar of lead. Coarse fodder must be avoided.

RED EPIZOOTIC DISEASES OF THE PIG.

Swine Plague (Schweineseuche): Contagious Pneumonia:
Hog Cholera.

GENERALITIES ON RED EPIZOOTICS OF THE PIG. For a long time little was known of the pathology of infectious diseases of the pig. Of late years the researches of Eggeling, Löffler, Schütz, Lydtin, Schottelius, etc., have enabled us to recognize therein several well-characterized epizootic diseases from an etiological, symptomatic, and anatomo-pathological standpoint. Until 1880 all epizootics of the pig were designated under the collective name of swine plague; but bacteriological researches have shown that under that denomination have been described:

1. *Swine plague*, a specific septicemia produced by a bacillus which is very small and characterized, from an anatomical standpoint, by a gastro-enteritis and a hemorrhagic nephritis, by a tumefaction of the spleen, and a parenchymatous inflammation of the liver, heart, and voluntary muscles.

2. *Contagious or infectious pneumonia*, pleuro-pneumonia, which is produced by ovoid bacteria; it is remarkable for its tendency to pulmonary necrosis and to caseous lesions.

3. *Hog cholera*, a diphtheric inflammation of the large intestine, with a participation in the process of the lymphatic ganglions of the neighborhood, which are the seat of intense alterations. It is often complicated by pneumonia.

These diseases are not equally distributed in all countries: in Germany we find especially swine plague and infectious pneumonia; in America, England, Sweden, and Denmark hog cholera causes most damage.

Swine Plague (Schweineseuche: Rouget du Porc).

HISTORY. Under the name of swine plague (Schweineseuche) (epizootic of pigs, typhus of pigs, angina, red disease, Saint Anthony's fire, petechial fever, spotted fever, etc.) have been designated a whole group of diseases having as a common symptom redness of the skin. But in the porcine species a large number of external and internal diseases are accompanied by redness of the integument; thus the expression "redness" is very comprehensive and vague.

An attentive study of ancient literature shows that this expression is generally applied to *swine plague*, and, in the second place, to the disease which Löffler, Schütz, Lydtin, and Schottelius have called *epizootic disease of the pig* (*Schweineseuche*). Under the name of swine plague they have also described urticaria, erysipelatous inflammations of the skin, certain septicemic diseases, several poisonings, perhaps also helminthiasis (*Strongylus paradoxus*), heat-stroke, asphyxia, several internal and external diseases which are accompanied by redness of the skin (pneumonia), and very probably unknown infectious diseases.

Very divergent opinions have been expressed on the nature of swine plague. Omitting the theories which consider it as a poisoning by solanine (Bergmann), or as an infection determined by the agents of the typhus of man—theories which have attracted but few supporters from the end of the last century (Chabert, Viborg, Erxleben) to the year 1860—swine plague has generally been likened to anthrax. The denominations of “swine plague anthrax” and “anthrax fever” date from that period. This doctrine was combated toward 1856 by Nicklas and a few other veterinarians: experience has taught that the consumption of meat coming from pigs affected by this so-called anthrax never leads to unfavorable consequences in man. The discovery of the bacteridium by Brauel (1865) gave it a decisive blow. This author recognized that the pig generally resists inoculations of the virus of anthrax; he has never found the bacteridium in the blood of this animal. Harms (1869) has also described swine plague as an infectious disease which has no relation to anthrax, and consisting essentially of a serious gastro-intestinal affection, with parenchymatous alterations of the kidneys, liver, muscles, and lymphatic ganglions, etc., an affection in which the lungs do not at all participate.

In 1882 Eggeling showed that the generic name of “swine plague” served to designate the following diseases: 1. *sporadic swine plague of the head*, an infectious disease of wounds which is similar to erysipelas of the head in man; it leads frequently to gangrenous destruction of the inflamed regions; 2, *sporadic urticaria* with benign course; 3, *red infectious disease* (a disease that may be compared with scarlatina of man), which is marked by an acute inoculable exanthema and is characterized by copper-red spots of the integument on the surface of the larynx, between the front legs, on the abdomen, on the inner fascia of the legs, by inflamma-

tion of the respiratory mucous membrane, by pulmonary œdema, by alterations of the liver, kidneys, etc. ; 4, *enzootic disease of pigs* (*Schweineseuche*), the most frequent and most dangerous of all specific diseases of the porcine species ; it is a variety of septicemia which is accompanied by serious general symptoms, depression of strength, intense fever, a diffuse redness of the skin which appears at first upon the posterior regions and extends rapidly to the whole surface of the body ; its most important anatomical alterations are hemorrhagic gastro-enteritis and considerable tumefaction of the mesenteric ganglions. At the same date Löffler recognized in swine plague two distinct forms : true *swine plague* and *enzootic disease of the pig* (*Schweineseuche*). But the expressions adopted by this author do not correspond with those of Eggeling. Löffler's *swine plague* represents Eggeling's *epizootic disease*, while the *red infectious disease* of the latter is the morbid state designated by Löffler under the name of *enzootic disease* of the pig. Löffler's terminology has prevailed.

The researches of this bacteriologist have established that *swine plague* is produced by a very fine bacillus which has a striking analogy to that of septicemia of the mouse discovered by Koch.¹ If inoculated in the mouse, this bacillus is invariably fatal ; it is also very often fatal to the rabbit ; when this latter animal resists erysipelatous dermatitis produced by inoculation it possesses immunity. The *epizootic disease of the pig* is generated by small ovoid bacteria which much resemble those of the septicemia of Gaffky (rabbit) ; the cultures of these bacteria kill the mouse, guinea-pig, birds,² and the pig. It was in 1885 that Schütz found Löffler's bacillus in the spleen of a pig which had been imported from the Duchy of Baden ; later he observed it in pigs from the neighborhood of Berlin, and has given a detailed description of it. In the

¹ Authors generally attribute the discovery of the bacillus of swine plague to Pasteur and Thuillier, whose works antedated those of Löffler. Thuillier described it as an 8-shaped micro-organism, doubtless because he examined it in fresh blood without any coloration. Such is not, however, the opinion of all our bacteriologists. Concerning the question of priority, Cornil and Babes express themselves thus : " There is no doubt that Klein, Pasteur and Thuillier, Detmers, Baillet and Jolyet, and Cornevin had not seen the true microbe of swine plague of the pig, which is the bacillus that is described in Löffler's first observation and in Schütz's facts." (Cornil and Bates: *Les Bacteries*, 3d ed., Paris, 1890.)—N. D. T.

² The chicken is refractory to its effects, and the pigeon does not succumb when the infection takes place through the digestive canal. These cultures are also fatal for the rabbit.—N. D. T.

same year Schottelius also found this micro-organism in pigs coming from Baden. Swine plague is a disease which is well known at the present day ; this is due to the numerous observations of which it has been the subject of late years, to the inoculations made by Schottelius and Lydtin in the Grand Duchy of Baden, and to the researches of Schütz on the etiology, pathology, symptomatology, anatomical alterations, and prophylaxis of the disease. The works of Pasteur and Thuillier, also of Cornevin, have contributed much to clear up its nature.¹

Bacteriology. Löffler, Schütz, and Schottelius have described the micro-organisms of swine plague. These are very thin bacilli, which are 0.6μ – 1.8μ in length, and can only be seen by means of high powers of the microscope (immersion, Abbé's condenser). They exist in the blood, and consequently in the capillaries of all organs. They are found suspended in the plasma, mixed with red corpuscles, and also very frequently in the interior of the leucocytes. They are especially numerous in the spleen, kidneys, lymphatic ganglions, etc. Morphologically they closely resemble the bacilli of the septicemia of Koch, with which Schütz wished to identify them. The artificial mediums most favorable to their development are slightly alkaline bouillons, which are prepared with horse meat, beef, or pork and exposed to a temperature between $+36^{\circ}$ and $+15^{\circ}$ C. ; they are also easily cultivated in the blood-serum and in vitiated watery fluid ; but are not developed on potato,² in vegetable infusions, or in bouillon prepared with meat obtained from pigs which were affected by the disease (Schottelius). They are anaërobic ;³ they swarm therefore in the deep layers of the mediums where they are made to vegetate. Cultures made by pricking take a corniferous aspect or that of a bottle brush (Schottelius). It is probable that these bacilli do not generate any spores (the opinion of Schottelius, who admits their sporulation, has been combated recently by Cornevin and Kitt ; these authors base their objections on the inactivity of the dried virus). Dampness seems to be necessary to

¹ The micro-organisms of swine plague and of pneumo-enteritis act differently in connection with staining fluids. The bacilli of swine plague are stained with Gram's and Weigert's fluids; the bacteria of pneumo-enteritis are not stained by these two processes.—N. D. T.

² When protected from air they may vegetate on the potato; but the culture is quite difficult and restricted.—N. D. T.

³ They are also aërobic; nevertheless, they vegetate much better in a vacuum or surrounded by an inert gas than when exposed to the air.—N. D. T.

the preservation of the vitality of the bacilli, which, according to Kitt, resists putrefaction.

Recent researches have shown the influence exerted on them by the various causes of destruction. They are killed within from fifty to eighty hours by a slow desiccation, in twenty minutes by water at a temperature of 46° C., and in two minutes by this same liquid raised to a temperature of 90° C.; dry and hot air (46° C.) kills them in three and a half hours; they do not resist so long higher temperatures (60° and above). Intense cold (-3° to -8° C.) kills them in thirteen days. They are also destroyed by quicklime, chloride of lime, and hot lye. In salted meat they die within a month. Saturated solutions of boric or gallic acid, arsenic, benzine, chloride of zinc, phenicated alcohol, acting for forty-eight hours, do not destroy their vitality. In sea water they lose their virulence in twelve days; they have been found there still living after twenty-four hours.

If we inoculate the bacillus of swine plague in the white mouse it dies, producing the symptoms of septicemia. Kitt has recognized that the white mouse and field mouse are quite differently affected by swine plague, as in septicemia: while the first dies from inoculation of these diseases, the field mouse possesses immunity from them. In the rabbit inoculated swine plague sometimes determines death within five to nine days, sometimes only a local cutaneous disease and immunity. The inoculated pig dies with symptoms characteristic of swine plague;¹ at the autopsy we find bacilli in all the organs. The pigeon and white rat also die. The virus is inactive in the guinea-pig, donkey, horse, ox, and sheep (Cornevin, Kitt). These animals must be considered to possess immunity. From these facts we must conclude that swine plague has nothing in common with chicken cholera, and that it probably does not affect animals of the bovine species (see Preusse's observations).

Pathology. The digestive mucous membrane is the principal mode of entrance of the bacilli. Cornevin has well established this fact, previously admitted by Pasteur, Schütz, Schottelius, and Lyd-tin; he has shown, besides, that even in cases where the disease is inoculated into the subcutaneous tissues the intestinal canal contains specific micro-organisms which reach this region through the channels of circulation and by crossing the vascular walls. These facts

¹ Death occurs in only a certain number of cases; young pigs resist much better than adults.—N. D. T.

have been confirmed by Kitt. It is probable that the infectious elements can only be introduced into the intestinal mucous membrane by means of a wound, or a solution of apparent or concealed continuity. Once in the blood they multiply rapidly; a very active pullulation also takes place immediately after death. They exert their noxious action by obstructing the capillaries or in generating a ptomainic poison (Schütz), the effects of which are particularly felt by the nervous and muscular systems and by the parenchymatous cells of the large glands (cerebral and paralytic symptoms, anatomical alterations of the glands.

It does not appear that infection is produced through the pulmonary organs; the bacilli, in fact, are killed by desiccation.

In the rabbit the virulence of swine plague becomes attenuated.¹ The process which is used by Pasteur in order to weaken the virus is based on this fact. Kitt has remarked that after one passage through the rabbit the activity of infectious elements is sufficiently attenuated within five or six days to allow a vaccination of the pig with blood or exudate which is collected at the point of inoculation. The attenuated virulence of the bacilli is fixed and permanent, and the pig acquires or may acquire immunity by a single inoculation of these agents. Preventive inoculation could thus be practised at one time, which would lessen the danger of contamination by inoculated subjects.

AFFECTED ANIMALS AND WAYS OF PROPAGATION. Swine plague is a very common disease which exists in an enzootic or epizootic state in most countries of Europe. It is stationary in certain districts. In Bavaria it was formerly limited to the region of the Danube; in the southern part of this country it was absolutely unknown (Kitt). The bacillus exists in different mediums, but particularly in stagnant waters; in Berlin, Löffler has found it in the water of the Pauke;² he found conditions favorable to its perpetuation in the valleys and low plains with slow running streams, in clayey, strong and damp earth (Lydtin); sandy and granitic soils are much less favorable mediums for it. Swine plague is specially prevalent during the warm season; in winter we observe but isolated cases; hot seasons and storms seem to favor its development. In the

¹ In passing through the rabbit the virulence increases for this animal, and is attenuated for the pig.—N. D. T.

² The Pauke is a little rivulet which runs through the park of the veterinary school and flows into the river Spree.—N. D. T.

Duchy of Baden it claims numerous victims on the small farms and in establishments where the pigs are fed on residues of distilleries. As predisposing etiological circumstances, damp, badly-ventilated and badly-kept stables, also alimentation with tainted substances, have always been pointed out, and were formerly considered, as direct causes of the trouble.

During the first months of life the predisposition to swine plague is least evident. It has a marked predilection for adult subjects (of three to twelve months); animals between the second and third years of life, however, are not spared; it is rare in older animals. Suckling pigs may consume the milk of diseased sows without danger, although, according to Konbasoff, the bacilli sometimes pass into this liquid. We have known for a long time that the degree of contagion of the pig for swine plague varies with the breed. In Austria Hable has seen animals of imported breeds affected in much larger numbers than those of domestic breeds. In Baden it is in the Chinese and English breeds, either pure or crossed (Suffolk), that the disease claims most victims; the Yorkshire, which are either pure or crossed, die in only small numbers. The most resistant animal is the domestic pig, which, consequently, is not suited for inoculation experiments.

The disease is propagated by direct contamination; but cases occur where it seems to be of miasmatic origin. Its epizootic prevalence in some regions renders the exogenous development of the contaminating factor extremely probable. Contamination is mostly produced by the ingestion of infected excrements and of tissues of animals which are affected or have died of swine plague. No facts have been observed establishing transmission by the intermediation of air. The animals in pens located in the neighborhood of those where the trouble exists are never contaminated in that way. Contagium is always fixed. (Buchner, who relies on recent experiments, admits that swine plague may be developed by the inhalation of infectious agents which penetrate through the intact pulmonary mucous membrane.)

Swine plague is disseminated by shipping the meat of diseased animals, and especially by retailing it from village to village. This was observed by Wirth toward 1840, and has been confirmed since by a large number of observers (Bleigenstorfer, Ebersbach, Fünfstück, Toelsch, Bühler, Zschokke, Lydtin). Hitt has demonstrated the truth of this theory by experimentation. Residues of

slaughter-houses and kitchens, the water in which the meat has been washed and the utensils are the principal propagators of the disease. Lydtin has seen it invade successively pig-pens which were situated along a rivulet into which ran the washings of a locality where the trouble existed. It is often propagated to great distances by pigs which are driven in flocks on the highways, as was observed by Grimm in 1860. Finally, it has sometimes its source in the ingestion of pieces of cadavers of pigs, mice, or infected white rats (Lydtin). According to Cornevin, it may also be produced by the ingestion of the brine of diseased meat.

A first infection generally confers immunity. Recurrences are extremely rare (Lydtin).

Symptoms of swine plague. After a period of incubation of three days at least, the disease appears suddenly, without any prodromic phenomena, and is marked from the onset by alarming symptoms. The animals refuse all food and bury themselves in their litter; the fever is very intense, defecation is delayed, the temperature reaches 43° C.; there are nausea, vomiting, and serious nervous troubles: extreme weakness, drowsiness, stupefaction, apathy, and paralytic conditions of the hindquarters. Sometimes also we observe muscular spasms and grinding of the teeth. The conjunctiva is dark-red or reddish-brown; at certain times the eyelids are closed. In the regions where the skin is thin (abdomen, umbilical and lower pectoral regions, perineum, inner fascia of the thighs, ears, and shoulders) we remark from the beginning, or after a few days only, spots of the size of a hand, which are at first clear red, afterward dark red, and finally bluish; when confluent, they form irregular surfaces, the color of which is brownish or reddish-blue. They are neither painful nor prominent on the surface of the skin; in a few cases we observe a slight inflammatory tumefaction. On the intense red-colored spots, especially on those of the ears; at times the skin is covered with vesicles, at other times it is gangrenous. In cases of subacute evolution the redness is slight; sometimes it appears shortly before death or immediately after; it may be entirely wanting; in other instances it extends over the whole surface of the body. Its redness is due to a serious hyperemia produced by a rapidly-increasing weakness of the heart, and to a decomposition of red corpuscles (elimination of the coloring matter). Soon the excrements become diarrheic, mucous, and at times bloody; finally the respiration is considerably accelerated,

and a general cyanosis appears (pulmonary oedema). Death is produced by exhaustion and a falling of temperature (37° C. and below); it occurs ordinarily on the third or fourth day. When the disease has a very rapid course it may end fatally in twenty-four hours. It lasts rarely more than one week.

Under the name of chronic swine plague Cornevin, Hess, and others have described a morbid state which at times succeeds acute swine plague. Thus also does the clinical picture of *chronic swine plague* differ considerably from that of the acute form. The appetite is preserved, but the animals lose flesh and become gradually weaker; diarrhea is continuous, the abdomen is contracted, and the breathing is very short, etc.; consumption is soon marked. Within a few months the symptoms of scurvy appear or decaying of the hair (intra-bulbar hemorrhages), dropping of the hair, hemorrhages and ulcerations of the gums. May not scurvy and decay of the hair be chronic forms of swine plague? Besides these symptoms (and preferably in inoculated subjects) we notice localizations on certain organs (enteritis, verrucose endocarditis, fungous arthritis). In all these chronic states red spots were observed upon the skin in the beginning of the affection, at the time of exacerbations, and immediately before death.

Prognosis. In the large majority of cases the patients die. The average mortality is from 50 to 85 per cent. In Bavaria, according to Lydtin, it varies from 50 to 75 per cent. In Denmark and Schleswig it reached 87 per cent. in 1873. These figures sufficiently indicate that the prognosis is always very serious. In many countries swine plague causes enormous losses. In the Duchy of Baden for a period of ten years its damages have amounted to 1,875,000 francs, and for the year 1884 to 336,000 francs; the number of sick was 6000 in 1875 and 4000 in 1876. In Switzerland the money loss amounts to hundreds of thousands of francs (Zschokke and Hess). The damage caused by it in Brittany is estimated at 3,000,000 francs. For the German Empire the sanitary service rates at nearly 6,500,000 francs the damage caused annually by swine plague. The gravity of the prognosis is lessened when the disease is prolonged for more than four days; when that period is passed the chances are in favor of a recovery from the trouble.

Anatomical alterations. The autopsy shows lesions of septicemia: a general infection without marked alterations of a particular organ, obstruction of the spleen, hemorrhagic or diphtheric

gastro-enteritis, a considerable tumefaction of all the lymphatic ganglions, a hemorrhagic or parenchymatous nephritis, parenchymatous hepatitis, myocarditis, and myositis. The description given of it by Schottelius is essentially the same as that of Schütz.

1. The obstruction of the spleen is the consequence of an intense hyperemia with abundant proliferation of the parenchymatous elements (neoformations of splenic elements). The organ is hypertrophied but not softened, as in anthrax; it is, on the contrary, resistant by reason of the tension of its capsule. The splenic pulp is soft, reddish-blue on section; we do not recognize any hemorrhages therein.

2. The gastro-intestinal mucous membrane is the seat of an acute hemorrhagic phlegmasia. The gastric mucous membrane is at times of a uniform dark red and sometimes marbled; it is tumefied, of suspicious aspect, rugous, irregular, and coated with a layer of viscous mucus and strewn with superficial eschars. The glands are inflamed (glandular gastritis). The intestinal mucous membrane is tumefied, especially on the upper edge of the folds, in the contracted portions of the intestinal grêle, and in the neighborhood of Peyer's patches; it is covered with reddish mucus and studded with hemorrhages and superficial eschars; more rarely we observe therein (in the cæcum and large colon) circumscribed diphtheric alterations.

3. The solitary follicles and Peyer's patches are inflamed; their surface is rendered irregular by small prominences, which vary in size from a millet seed to that of a lentil; sometimes these organs are overrun with hemorrhages and surrounded by a red zone. Schottelius has frequently found them escharified or ulcerated; he has pointed out the similarity of these alterations to those of typhoid fever of man. Johnes has also found ulcerations in all serious cases. The mesenteric ganglions are tumefied, dark red, and softened; their section has a reddish-gray ground, which is clouded by dark red spots. The periganglionic tissues are hyperemic and overrun with hemorrhages.

4. Hemorrhagic nephritis is indicated by an increase in the size of the kidneys and by a reddish-gray coloration of their surface; the medulla is dark red; the cortical substance is hypertrophied and overrun with hemorrhages. In mild cases we observe but a parenchymatous inflammation of the kidneys (turbid tumefaction). This lesion is often accompanied by catarrhal nephritis.

5. Parenchymatous nephritis is marked by a turbid tumefaction and increased dimensions of the liver, by a brownish-gray coloration of the section and hypertrophy of the *acini*.

The striated muscles show a gray coloration; they are watery, shiny, soft, flabby, crooked-like, and sometimes strewn with hemorrhages. The myocardium is marked with similar alterations and sub-endocardiac ecchymoses.

In the abdominal, pectoral, and pericardial cavities we find small quantities of clear reddish-yellow serum, which is more or less flaky. Under the serous membranes, especially under the pericardium and on the surface of the auricles, we observe punctiform hemorrhagic centres. Abundant hemorrhages in the brain and dorsal and lumbar portions of the spinal cord (Cornevin) are rare. The red coloration of cutaneous patches is less intense after death.

The lungs are sometimes unaltered, and sometimes œdematous (*post-mortem* œdema). Finally, on microscopical examination we find in the blood, no matter from what organ it may be obtained, but more particularly in the spleen and kidneys, the characteristic bacilli.

Diagnosis. At the autopsy the diagnosis of swine plague is based essentially upon the tumefaction of the spleen, gastro-enteritis, alterations of the lymphatic apparatus, and upon the absence of inflammatory pulmonary alterations. In doubtful cases we must resort to inoculation. Schütz advises the employment of the white mouse as an agent. The operation is very simple: it consists of depositing in the subcutaneous connective tissue of the dorsal region a few drops of blood or very small fragments of splenic tissue. If we have really to do with swine plague, the inoculated subjects become sick within twenty-four hours; they are depressed, the hair is erect, the back arched, and breathing very accelerated; a mucous, gelatiniform exudate escapes from the lachrymal sac; death occurs on the fourth day.¹ Johné advises resort to cultures by puncturation. The manner of operating is as follows: the spleen is washed in a 1 per cent. solution of subli-

¹ In order to differentiate *swine plague* and *pneumo-enteritis* we must inoculate at the same time with medium or weak doses of virus a pigeon and a guinea-pig. If the latter resists and the first one dies, we have swine plague; if, on the contrary, the pigeon remains unharmed, and if the guinea-pig dies, we have a case of *pneumo-enteritis*.—N. D. T.

mate; then, by means of an incision made on Glisson's capsule toward the centre of the organ, we introduce into the splenic pulp, at a depth of 4 to 5 centimetres, a platinum wire which has been first sterilized and afterward plunged to the same depth into peptonized gelatin, taking care to uncork the tube which contains it only at the moment of operating, and to hold it inclined with the opening turned downward. This tube is then closed again and placed in the ovum. Within four or five days, if we have a true case of swine plague, we see the development of a bottle-brush-shaped culture which is altogether characteristic.

Differential diagnosis. The diseases which may be confounded with swine plague are:

1. *The infectious disease of the pig (infectious pneumonia)* is distinctly distinguished from swine plague by its localization in the lungs. Here we have as differential symptoms troubles of breathing, an inflammatory tumefaction of the red patches of the skin, and the result of the bacteriological examination (ovoid bacteria).

2. *Urticaria.* As a rule, this disease is very benign, and never occasions the serious nervous troubles observed during the course of swine plague. The temperature is not very high, and, in general, the appetite is preserved. While in urticaria there exist cutaneous patches of a dark color, these are of special pathognomonic character; they are not blotches due to a common passive hyperemia of the skin, but spots the dimensions of which are nearly that of a silver dollar. These blotches, which are formed by a hemorrhagic infiltration of the skin, do not disappear under pressure of the finger; only rarely do they become confluent, and after death take a rhomboidal form.

3. *True erysipelas* is a complication of traumatisms, and is especially observed in wounds of the head. It is characterized by a painful cutaneous phlegmasia, which frequently ends in gangrene (mortification of fragments of skin along the edge of the ears).

4. *Heat-stroke* is an apoplectiform disease to which very fat pigs which are transported during very intense heat are particularly exposed. It is marked by a reddish-blue coloration of the skin, which is due to blood stagnation. At the autopsy lesions of suffocation are found.

5. *Anthrax* is very rare in pigs, where it generally takes the form of glossanthrax. We observe dysphagia or dyspnoea and a carbuncular tumefaction of the skin of the laryngeal region. A recog-

nition of bacteridia, the length of which is nearly ten times that of the bacillus of swine plague, insures the diagnosis.

Swine plague may be confounded also with traumatic erythema. In pigs which are driven some distance it is not rare to observe erythema produced by the action of traumatisms, and in sows during the period of lactation we see these quite frequently on the mammæ, where it is produced by repeated irritations resulting from sucking.

Treatment. When swine plague takes a rapid course it is rare that intervention is prompt enough to save the patients. Formerly it was customary to administer an emetic in the beginning. All authors are unanimous in recognizing the advantages that this treatment affords. Berner reports that in 1858 it produced a cure in three-fourths of his cases. The salutary influence which is produced by emetics is evidently due to the ejection of infectious matter. In cases where treatment may subdue the trouble large doses of calomel (three to five grammes) ought to be of advantage, because they effect a fairly complete disinfection of the intestinal canal, due to the formation of sublimate.

But the prophylactic is much more important than the curative treatment. Healthy animals must be immediately separated from the sick and placed in a special stable. The pig-pens should be thoroughly disinfected; it is especially important to destroy the noxious effects of the excrements. According to researches made by direction of the German Sanitary Service, quicklime, hot lye, and chloride of lime are sufficient for disinfection. When we buy pigs of unknown origin it is prudent to subject them to a quarantine before installing them in the common stable. We must preserve healthy animals from all contact with meat or refuse of any kind coming from pigs affected with swine plague. A regular inspection of meat for sale is also an excellent measure. Finally, it is desirable that swine plague should be the subject of legal regulations similar to those existing in Denmark and England for a long time and in Austria and Switzerland since 1866. The following are the principal sanitary measures which are applied in Austria (law of April 10, 1886): The cadavers of subjects which have died of swine plague must be destroyed; the consumption of meat from pigs affected by swine plague is only tolerated in the locality where the epizootic exists; when the epizootic makes its appearance during transportation the train must be stopped and

placed in quarantine as soon as the trouble is recognized. In the German Empire it is probable that an obligatory declaration, which is regulated by articles 9 and 10 of the law of June 23, 1880, would also be applied to swine plague, and that it should be combated by energetic sanitary measures.

PREVENTIVE INOCULATION OF PASTEUR. Pasteurian inoculation has lately taken first place in the prophylaxis of swine plague. While quite a considerable number of pigs have been inoculated in different countries (France, Germany, Switzerland), we cannot yet express a final opinion on the value of this method.

1. In the Duchy of Baden, in 1885, there were inoculated under the direction of Lydtin 237 pigs, from nine to nineteen weeks old, of a weight varying from 11 to 47 kilogrammes, and belonging to eight different races. These animals were distributed in fifteen stations, embracing the whole country. They had taken into account the climatic and geological circumstances, the regimen and hygienic conditions, as well as the infection of the localities. The inoculations were performed by one of Pasteur's assistants. The manner of operation is very simple. The pig is placed upon its back, the posterior members are spread apart, and we inject under the skin of the inner fascia of the thigh part of the contents of a Pravaz's syringe (about one-tenth of a cubic centimetre). Out of 237 pigs 119 were inoculated; the other 118 animals were not inoculated. Inoculation was performed in two series: the first time with the weak lymph (first virus), and twelve days later with the strong lymph (second virus). After another period of twelve days 60 animals of each class were taken and subjected to the action of the contagium, either by inoculation with the strong virus or by forcing them to ingest cadaveric refuse of animals which had died of swine plague. The results obtained were the following:

Out of the 119 pigs inoculated 96 (80 per cent.) manifested certain morbid phenomena (elevation of temperature) after the first inoculation, 18 (15 per cent.) presented all the symptoms of swine plague, 6 (5 per cent.) died. After the second inoculation, among the 113 remaining animals 46 per cent. showed a marked hyperemia, 7 per cent. were manifestly affected by swine plague, but none died. Of the 118 check animals 62 per cent. perished after the first inoculation (fever); only 1 died of swine plague as a consequence of the second inoculation; 36 per cent. were contaminated

by the inoculated animals (fever); 4 showed the symptoms of swine plague, and only 1 died. Of the 60 pigs which were inoculated and previously inoculated with strong virus, 19 had a slight fever and 4 were slightly sick; of the 60 non-inoculated pigs which were inoculated with strong virus, 37 (61 per cent.) were quite seriously sick, 24 (40 per cent.) were affected by swine plague, and 16 (26 per cent.) perished.

Out of 462 pigs which were inoculated in the Duchy of Baden in 1886, 26 (5.6 per cent.) died; in 1887, of 152, only 2 (1.3 per cent.) died.

These figures establish that Pasteurian inoculation gives to the animals a certain immunity, but it is not without dangers: the inoculated subjects may contaminate the healthy animals, and thus propagate the disease; moreover, it produces certain morbid phenomena and sometimes death (4 per cent., according to the preceding statistics). Supposing that all the animals of one country were inoculated, and that the proportion of the cases of death was 4 per cent., inoculation would occasion greater losses than swine plague itself. The general mortality caused by the disease is in fact only 2 per cent. for the Duchy of Baden.

2. In Switzerland Hess inoculated 46 pigs with Pasteur's lymph. After the first inoculation 11 were seriously sick, 3 had to be slaughtered, 6 were affected by consumption, and only 2 were cured. After the second inoculation no acute accident occurred, but later 9 pigs became sick and showed at the autopsy intestinal, endocardiac, and articular lesions. Of the 46 pigs inoculated, 26 did not present any disturbance, the other 20 were sick, and in 15 of these the disease persisted in a chronic state. Hess and Guillebeau admit that these conditions consecutive to inoculation were produced by impurities contained in the inoculated lymph;¹ they acknowledge that their confidence is weakened. Hess has observed, moreover, that old and very fat animals withstand inoculation very badly. The latter, when performed on animals older than four months, is said to be a hazardous operation.

3. In France a large number of inoculations have been made

¹ This opinion is the most likely. Quite recently one of us has inoculated, conforming strictly to the indications given by Pasteur, 50 young pigs belonging to twenty-one owners, of three different localities. Two months later none of the inoculated animals had died, notwithstanding that swine plague continued to exist in the district.—N. D. T.

with variable results. Of 35 pigs inoculated by Eloire only 22 withstood the operation. Herbet, during the early period when he first used inoculation, saw almost all the animals die, but later he was able to inoculate 4000 subjects without having to record any direct losses. Cagny has obtained very good results.

4. In Lorraine, during the year 1885, Dieudonné inoculated 21 pigs, which all remained unharmed; in 1886 he inoculated 283 subjects with a loss of 2.1 per cent.

5. In Prussia inoculations have been performed by Schütz and Jacobi (Obornik). In old pigs the losses were considerable (50 per cent.). But in 226 young pigs, which were less than sixteen weeks old, only 3 (1.3 per cent.) died. The effects of inoculation here have been very favorable.

From a general review of these observations it results that Pasteurian inoculation does not constitute at the present time a prophylactic method which is applicable in practice. But it is open to improvement. The experiences of Schütz have shown that it is also efficient when we operate with a lymph which has passed successively through several organisms. Kitt thinks that we should be able to confer protection in a single inoculation.¹

¹ In France, during the year 1887, swine plague existed in 39 departments, and caused losses which were estimated at 500,000 francs. Out of 18,815 pigs which were affected, 15,328 died. The Pasteur Institute has furnished virus for 12,280 animals. The results of inoculation have been known in only 7467 cases: 57 died after a first inoculation, 10 after the second, and 23 during the rest of the year; that is to say, a total loss of 90 (12 per cent.). The mortality after the inoculation period was only 3 per cent. (Tisseraud: "Rapport sur le Service des Epizooties en 1887," in *Receuil Vét.*, 1889.)

Revel, veterinarian of the Department (province) of Aveyron, has just published the favorable results which inoculation gave him during the years 1885-86. Of 530 pigs inoculated in 1885, 7 died between the first and second inoculations; all the others were resistant; in the localities where these inoculations were made, 556 pigs which were not inoculated died from swine plague. In 1886, 502 pigs were inoculated; 12 died from swine plague between the first and second inoculations; none of the surviving animals died from that infection, which has, however, claimed more than 500 victims in the region. The age of four months, which is assigned by Pasteur as the extreme limit in order to perform inoculation, may be exceeded; this operation is only dangerous for animals that are more than eight months old. The average duration of immunity has been about one year. (Revel: *Le Rouget du Porc et son Traitement*, Paris, 1891.)

In Hungary, in 1889, out of 15,057 pigs which were inoculated in forty-two towns none died of swine plague in the course of the year. In 1890, 249,800 pigs were inoculated. For 119,900 the sanitary reports have published the following results: 297 (0.24 per cent.) died after the first inoculation; 134 (0.11 per cent.) after the second, and 1397 during the rest of the year; a total loss of 1828 subjects (1.53 per cent.). (Hutyra: *Jahresber. über das Veterinärwesen in Ungarn*, Budapest, 1890.)

In France, swine plague is known under the name of *rouget* or *mal rouge*. In 1882 Pasteur and Thuillier described as the agent of this disease an 8-shaped microbe (diplococcus), which presents a great analogy to the bacillus of chicken cholera (this diplococcus is perhaps the ovoid bacterium of the epizootic disease of the pig of Löffler and Schütz(?)). In 1883 Pasteur prepared a lymph destined for preventive inoculation; since that time he has improved his process. Pasteur's lymph does not contain the 8-shaped bacterium; but Schütz and Schottelius have found the fine bacillus of swine plague in it. It is probable that Pasteur, in the beginning of his studies on the epizootic diseases of the pig, had to deal with infectious pneumonia. The process of preparation of the lymph is not exactly known; we know only that the latter is obtained by a series of transitions through the rabbit and by culture *in vitro* (tube). According to Pasteur, the inoculated animals are only subject to slight troubles, and acquire immunity from swine plague. Pigs that are from nine to sixteen weeks old are inoculated the first time with the weak lymph (first virus), and ten to twelve days later with a stronger lymph (second virus). The inoculation is made into the subcutaneous connective tissue. Pasteur has transmitted the disease to the rabbit, the pigeon, and sheep; he has also infected the pig through the digestive organs. His first inoculations were made in the Department of Vaucluse in 1882. The ameliorated lymph has been furnished since 1884. Baillet and Jolyet have also cultivated the bacillus of swine plague.

Epizootic or Infectious Pneumonia of Swine Plague.

(*Schweineseuche*.)

Bacteriology. The infectious agent of contagious pneumonia of the pig is an ovoid bacterium, which is from 1–2 μ long, and from 0.4–0.5 μ wide (Löffler and Schutz); it is colored upon both extremities, and is multiplied by segmentation. It has a great similarity to the microbe of septicemia of the rabbit of Gaffky (Schütz admits the identity of these micro-organisms); it resembles also the bacillus of chicken cholera. The cultures made by puncture in peptonized gelatin show along the tract numerous small white spots, which are confounded by a whitish-gray layer with punctiform peripheric centres; on the surface there is produced around the puncture a whitish-gray swelling; the gelatin is not

liquefied. On coagulated serum the cultures have a variegated aspect. The inoculations in the subcutaneous connective tissue kill the mouse and the rabbit within forty-eight hours; the blood and purulent fluids contain the characteristic bacteria. The guinea-pig, pigeon, and rat are hard to infect. The chicken is absolutely immune. Two pigs which were inoculated died within twenty-four to forty-eight hours. At the place where the inoculation had been made we recognized a carbuncular inflammatory oedema; the pathologic elements existed in large numbers in the whole organism; with their culture the disease has been reproduced. A pig in which immunity had been conferred against swine plague died from infectious pneumonia sixty hours after the inoculation of the ovoid bacterium.

The infectious agents are very numerous in the gangrenous centres of the lungs, in the pleuritic exudate, and in the bronchial ganglions; they are more rarely found in the blood and organs of the abdominal cavity.

The injection of cultures into the lungs through the pectoral walls determines there a gangrenous phlegmasia with necrotic lesions of contagious pneumonia of the pig. Schütz has produced the disease experimentally by the inhalation of dried-up cultures. Infection by the digestive organs has also given positive results.

The specific bacteria, which are considered by Schütz as identical with those of septicemia of the rabbit, present, according to Kitt, a striking analogy to the microbes which he has found in the *epizootic disease of wild animals* (*Wildseuche*). He believes in the identity of these two diseases. Quite recently Hüppe has recognized this theory;¹ he admits besides that the bacillus of chicken cholera is similar to that of contagious pneumonia of the pig, and that the affection of contagious pneumonia of the horse must also be related to it. But that is an opinion which is not as yet based upon any strictly established fact.

Pathology. In comparison with *fixed* contagium of swine plague, that of infectious pneumonia seems to be very subtle. According to Schütz, in most cases the infection is produced by inhaled air; the disease is therefore extremely contagious. Pigs which are driven in herds on the highways form the principal agents of its propagation; often, in fact, there exist chronic inflammatory centres in the lungs of animals which were previously affected. Schütz admits a

¹ Hüppe: Berlin. klin. Wochenschr., 1886.

possibility of infection by solutions of continuity of the skin and by the digestive mucous membrane. For this author the disease consists essentially of a necrotic infectious pneumonia which is accompanied by certain secondary alterations. Johne believes also that infection may be produced in several ways (skin, respiratory apparatus, intestine); but, according to him, the pulmonary disease would only represent a determination of the infectious process, which might also be marked by carbunculous manifestations of the skin; sometimes even it would not produce any localization, and the clinical picture of infectious pneumonia might be confounded with that of septicemia.

Anatomical alterations. The alterations are those of infectious pneumonia with multiple centres, and with general secondary infection (Schütz). In the lungs we find reddish-gray hepatized regions, in the centre of which exist yellow spots, which are distinctly circumscribed, and of the size of a "grain of sand." These spots, which are very numerous and correspond to small, necrosed islands, are formed by a discolored hemorrhagic inflammatory exudate; their periphery is bordered by a reddish-gray hepatized zone. The necrotic centres have often determined a pleurisy and a fibrinous pericarditis. The bronchial ganglions are tumefied. In the digestive apparatus we sometimes find a gastro-enteritis accompanied by lesions of the lymphatic organs. Among the general alterations we may mention parenchymatous degeneration of the liver, kidneys, heart, and spleen; in most cases the latter has preserved its normal dimensions. The lymphatic apparatus of the intestine and the mesenteric ganglions do not usually show any serious lesions.

In a few pigs Schütz has found caseous centres in the lungs, lymphatic ganglions, tonsils, bones, tendons, synovials, etc.; the alterations offer, then, a close analogy to those of tuberculosis, except the presence of the bacillus of Koch. These centres, which contain the specific bacteria, prove that the trouble may persist in a chronic state.

According to Schütz, the disease of pigs described by Roloff under the name of *caseous enteritis* might only be a form of the morbid processes which are generated by the ovoid bacteria (see Tuberculosis).

Symptoms. Infectious pneumonia, which is generally confounded with swine plague, is as yet quite imperfectly known

from a symptomatic standpoint. It takes usually a very rapid course; it often determines death within a few hours. It is marked by redness, tumefaction of the skin in the cervical region, and especially on the legs, by cough, dyspnœa, an intense fever, depression, and great weakness.

According to Hess, when the disease assumes an acute type its average duration is from three to nine hours. According to the same author, infectious pneumonia and swine plague coexist quite frequently in the same pen.

Schütz admits that the trouble may assume a chronic form (chronic pulmonary centres).

Treatment. From a prophylactic standpoint infectious pneumonia is combated by the same means as swine plague. Its very rapid course prevents in most cases any efficient therapeutic intervention.

Plague: Diphtheria: Hog Cholera: Swine Plague: Swine: Fever: Hog Fever.

HISTORY. In the last ten years authors have studied in England, America, Sweden, and Denmark a fatal disease of the pig which is given various names.¹

¹ In 1887 Cornil and Chantemesse studied at Chantilly a similar disease, which they have described under the name of *pneumo-enteritis*. "In the beginning the animals are tired and remain recumbent; at the same time cough appears, also a respiratory obstruction. The fever increases, the appetite is lessened, emaciation becomes more pronounced; the skin of the abdomen and flank often shows a reddish color, which has caused this affection to be confounded with swine plague; the skin of the neck shows blackish spots, which are due to an accumulation of dust and impurities, and on the surface of which the hairs fall or are easily pulled out. The patients lie down and remain quiet, and utter plaintive groans when they are displaced. From the beginning we observe a mucous, whitish, and fetid diarrhea, which sometimes persists to the end of the trouble; at other times it may be replaced by constipation. The total duration of the disease varies from twenty to thirty days. It is distinguished from swine plague by its slowness, by the predominance of pulmonary symptoms, and by the characters of the micro-organisms which produce it. All the exposed animals are sick, but some recover, and thereby acquire future immunity." At the autopsy we recognize as principal alterations nuclei of bronchopneumonia in both lungs and ulcerations of the large intestine. Cultures which are made upon gelatin, agar, or potatoes with lung or liver serum have been fertile. The cultures contained in a pure state small *oval bacteridia*, which were mobile, aerobic, anaerobic, and measuring $1\ \mu$ to $2\ \mu$ in length and $0.3\ \mu$ to $0.3\ \mu$ in diameter; it vegetated very well in an intermediary temperature from $+18^{\circ}$ to $+45^{\circ}$ C., without producing any spores. With these cultures the disease may be inoculated in the pig, rabbit, guinea-pig, and mouse. The pigeon has shown itself refractory to it.

The pathologic agent of *pneumo-enteritis* of Gentilly is also cultivated in distilled

1. In England Klein (of London) described (1878), under the name of *infectious pneumo-enteritis* (*hog plague*, *pig typhoid*, *hog cholera*, *swine fever*, etc.), an infectious disease in which the lungs, the serous membranes, and the intestine are principally affected, and which is produced by a specific bacillus. He cultivated the

water, where it may live for more than fifteen days; it resists congelation and desiccation, but is killed by a temperature of 58° C. acting for a quarter of an hour. Saturated watery solutions, iron, chloride of zinc, lime-water, picric acid, ammonia, phenol, sea-salt, pure essence of turpentine, sublimate at 1 per 1000, alone or added to hydrochloric acid at 5 per cent., biniodide of mercury at 5 per 2000, phenic acid at 25 per 1000, salicylic acid at 1 per cent., sulphuric, nitric, and hydrochloric acids at 1 per cent. do not destroy the virulence after one hour's contact. Of all the preparations which have been tried, the one that appears to be the most efficient is the following: water, 100 grammes; phenic acid, 4 grammes; hydrochloric acid, 2 grammes.

Cornil and Chantemesse have succeeded in attenuating the virulence of the elements of pneumo-enteritis by subjecting successive cultures to the action of air and heat (43° C.). At the end of ninety days the virus no longer kills the rabbit or guinea-pig, and it gives immunity to these animals. This attenuation of the virulence is fixed, and is transmissible to cultures made in series. Authors have tried to inoculate pigs. "Four pigs received successively at eight days' interval, under the skin, in a dose of 2 cubic centimetres, bouillon of virus culture which was ninety, seventy-four, fifty-four, and eight days old, and finally virulent virus. During the days following the inoculations the animals showed only a slight indisposition; two months after the beginning of the experiment they received in their food 1 litre of virulent culture in bouillon. . . . Two pigs which were not inoculated were subjected to the same treatment. Within 10 days these latter died with characteristic intestinal lesions. The inoculated animals did not show any important morbid phenomenon for two months; after this lapse of time two died. The autopsy showed intestinal lesions which were much developed and had a chronic course, and very slight pulmonary alterations. Both of the other inoculated animals resisted.

These first experiments showed that the pig acquires immunity from swine plague, with more difficulty than the subjects of other species.

In 1887 a very serious epizooty existed in the porcine species in the neighborhood of Marseilles—25,000 subjects died. This epizooty, which was studied by Fouque, Queirel, Rietsch, Jobert, Cornil, and Chantemesse, was determined by bacteria which presented but minute differences from those of the epizooty of Gentilly (see Cornil and Babes: *Les Bacteries*, 3d édit., Paris, 1890).

Galtier observed in the ovine species in the lower Alps an epizooty of pneumo-enteritis which was transmitted from the pig to the sheep. It was imported through pigs which had been recently bought, and has produced in sheep much more considerable losses than in the porcine species. The pathogenic agent found in the sheep was identical with that of pneumo-enteritis of the pig. By inoculation of cultures the author has transmitted the disease to guinea-pigs and rabbits; then he has retransmitted it to the pig and the sheep. He has also communicated it to the goat, dog, calf, donkey, and to poultry. It is transmitted from the mother to the fœtus. The inoculation of a goat in an advanced state of gestation has produced abortion. In the thoracic and abdominal cavities of the fœtus they found a sero-bloody discharge; this transudate and the blood contained the bacterium of pneumo-enteritis; their inoculation has reproduced the disease. (See art. *Epizootic Abortion*, and *Journal de Lyon*, 1889-90-91.)—N. D. T.

latter and transmitted the disease to the pig by inoculation (the rabbit and mouse also contracted the disease, while the pigeon resisted). In 1862 this disease appeared in England for the first time with marked intensity; its mortality often reached 75 per cent. In 1885 the number of sick pigs reached 40,000; 27,000 were slaughtered and 10,000 died. In 1886 it destroyed 35,000 animals. Klein recognizes a malignant and a benign form of this disease; both are extremely contagious. The bacillus, which is considered by this author as the pathogenic agent, is 1μ to 5μ in length, and $\frac{1}{4}\mu$ to $\frac{1}{3}\mu$ wide, with rounded extremities.

2. In North America, in 1877-78, the porcine species was decimated by a contagious disease that Detmers (of Chicago) designated by the expression *swine plague*; Law (of Cornell University) described it under the name of *hog fever*, and the farmers generally called it *hog cholera*. According to both these authors, the disease is said to have made its appearance in America nearly thirty-five years ago. It is extremely contagious, and is marked by the following symptoms: a dependent position of the head, cough, inappetence, rapid emaciation, great weakness, abnormal color of excrements, alternations of diarrhea and constipation, rosy spots on the belly, chest, and the inner fascia of the thighs and ears, etc. Its principal lesions are: pneumonia, pleurisy, pericarditis, peritonitis, alterations of lymphatic ganglions, ulcerative lesions of the large intestine, parenchymatous degeneration of the heart, etc. The microscope permits us to recognize in the fluids micro-organisms of various forms. Detmers designated one of these under the name of *Bacillus suis*, and called it the specific agent of the disease. The mortality of the latter has been 75 per cent. In the State of Nebraska 400,000 pigs (one-quarter of the whole number) died during the years 1884 and 1885. In 1885 the total loss for the United States was estimated at \$30,000,000. In Missouri the number of deaths was 200,000, and in Indiana 400,000 (one-fifth of the whole number). Salmon considers the benign form as a specific bronchial catarrh, and the serious form as a catarrhal pneumonia of the same nature. According to Law, the disease is transmissible from the pig to other species (sheep, rat), and reciprocally. Salmon has shown quite recently that the *hog cholera* of America is different from swine plague. The preventive inoculations of Pasteur have been recognized as inefficient; swine plague has a shorter period of incubation (three days instead of seven to fourteen)

and also a shorter duration (two days instead of eight or ten), and its mortality is less; the guinea-pig does not die from *hog cholera*, while the pigeon does; we very rarely find intestinal ulcerations in swine plague; they are almost constant in *hog cholera*. From a bacteriological standpoint the bacillus of swine plague is distinguished from that of *hog cholera* by a greater variety of form and development.¹

3. In 1887, *hog cholera* was introduced into Sweden (Schoonen, Södermanland, Stockholm, Gothenbourg), and from there to Denmark. In September, 1887, it killed a certain number of young pigs on the island of Amager and in some localities of the Danish coast; later it reached Copenhagen and the island of Seeland, where it was studied by Bang and Schütz, who recognized the *American* or *English disease*. Schütz gave to it the name of *hog cholera*.

No doubt exists either as to the identity of the diseases which were observed in England, America, Sweden, and Denmark, nor as to their dissimilarity with swine plague. Porcine plague greatly resembles the epizootic disease of pigs (*Schweineseuche*) which existed for a long time in certain regions of Germany. The bacteriological, anatomical, and clinical similarities which these morbid states offer must make us admit their identity; the differences which they show in their course and their manifestations are secondary, and due to multiple influences: period, climate, and localities.²

Pathology. The infectious agent of *hog cholera* usually enters with the food; the infection, therefore, especially takes place through the intestinal canal. The disease preferably exists in young pigs (up to the age of four months). The period of incubation varies from five days to three weeks.

Symptoms. *Hog cholera* may assume an acute or chronic form.

1. The first, which is ordinarily observed in the beginning of

¹ The bacillus of swine plague is immobile, it is well colored by the methods of Gram and Weigert; that of *hog cholera* is mobile, it does not take Gram's, and is poorly colored by Weigert's process.—N. D. T.

² The identity of epizootic diseases of the pig designated under the names of *hog cholera* (*cholera des porcs*), *swine plague* (*peste porcine*), *Schweineseuche* (*pneumonie infectieuse*), and *pneumo-enteritis* is not clearly demonstrated. *Hog cholera*, which is described by Salmon and other American authors, appears to be similar to *pneumo-enteritis* of Cornil and Chantemesse and to the epizootic disease observed in the neighborhood of Marseilles. Swine plague or swine pest is more closely related to Löffler's and Schütz's *Schweineseuche*.—N. D. T.

epizootic, generally develops in from five to eight days; but death may occur much more rapidly. The disease is indicated by inappetence and constipation; the animals, which are very weak, appear seriously affected—head and tail are hanging, the conjunctiva is injected; sometimes the eyelids are agglutinated by dried-up mucus; the temperature is increased (41° to 42° C.); the respiration is accelerated and laborious; in some cases we observe a nasal discharge, which is muco-purulent. Later, diarrhea occurs; the excrements are liquid and fetid, sometimes mixed with blood; diphtheric ulcerations are developed on the tongue (on the end, lateral edges, or lower fascia), on the mucous membrane of the cheeks, palate, tonsils; they generally present a grayish-white or yellowish-gray coloration; in many animals they have a gangrenous character. Quite often we observe red spots on the ears, groin, and the dependent part of the abdomen, on the inner fascia of the thighs, and in the neighborhood of the anus. Toward the end of the disease the animals lose control of the hind quarters; they can hardly stand, and remain almost constantly recumbent. Death is preceded by convulsions.

2. In the chronic form the symptoms are much less pronounced (the same peculiarity is noticed in *chronic infectious pneumonia* (*Schweineseuche*); the development of the patients is delayed; they remain small, sickly; they cough from time to time, and have diarrhea. We observe eczematous cutaneous eruption and a slightly marked redness of the ears.

Pathological anatomy. As prominent alterations we must mention multiple and circumscribed diphtheric centres of the mucous membrane of the large intestine (Schütz). They exist especially in the cæcum, on the surface of the point where the ileum joins it. These diphtheric centres are the result of a catarrhal or fibrinous inflammation of the mucous membrane. The exudate is formed of a dry, solid, grayish, whitish-gray, sometimes yellowish mass. The solitary follicles and Peyer's patches nearly always participate in the process; in the beginning they are tumefied, of a grayish-white color, and surrounded by a red zone; later they become gangrenous. Sloughs are found on the surface of the follicles, which gradually extend on the surface and in depth, and which at times involve the entire thickness of the intestinal walls, and are followed by elongated or rounded ulcerations. When several eschars become confluent large portions of the intestine are

transformed into dry or caseous patches, which are of a yellowish, brownish, or greenish color, and the thickness of which reaches at times from one-half to one centimetre; their surface is rugous and streaked with slight furrows; they are covered outside by the serous membrane only (necrosis of the intestinal wall); putrefaction renders them soft and friable; they become detached, leaving ulcerations which may perforate the intestine, and thus determine a peritonitis.

In serious cases we observe diphtheric alterations on the intestinal, stomachal, and buccal mucous membranes.

The mesenteric ganglions, which are greatly tumefied, frequently show lesions which are of tuberculous aspect. We find these in caseous centres surrounded by a connective-tissue envelope. In cases which have a chronic course the pulmonary regions are invaded by caseous processes, perforated and surrounded by wide, bluish, fibrous bands. The caseous centres which are located upon the periphery of the lungs produce an exudative pleurisy, which determines adhesions of the lungs to the costal walls, diaphragm, and pericardium.

The spleen is generally intact; sometimes, however, it is tumefied. The liver, kidneys, heart, and muscles are marked by parenchymatous alterations.

Treatment. The prophylaxis of hog cholera comprises the same indications as that of swine plague (sanitary police measures).

TYPHOID OF POULTRY.

Chicken Cholera: Chicken Plague.

HISTORY. Cholera and diphtheria are the two most serious infectious diseases of the chicken family. They are mentioned in the oldest works. Formerly and until a recent date *chicken plague* ("chicken death") was considered similar to cholera or typhus of man, from which we get the names *chicken cholera* and *typhus of poultry*. Some authors (Chabert, Hering) have considered it as a form of anthrax. According to Lemaistre, it made its first appearance in Lombardy in 1789 and existed in India in 1817. From 1825 it has been observed in France, where it occasioned great losses during the years 1830 and 1850, and especially since 1860. Toward 1830 it invaded Russia, Bohemia, and Austria. Lately it has been seen in nearly all the countries of Europe.

Long ago it attracted the attention of veterinarians, and, as a consequence, its symptoms, anatomical lesions, and mechanism of its propagation were perfectly known when its parasitic nature was discovered by bacteriologists. Benjamin, who, as early as 1851, admitted a contagium as the cause of the disease, remarked that men and dogs could consume the meat of affected birds with impunity. Delafond and others have observed its transmission to poultry and rabbits by the intermediation of the blood secretions and cadaveric remains; they have also recognized the virulence of the excrements and the important rôle that the latter play in contagion. Finally, it has been experimentally demonstrated that infection may be produced through the intestinal canal, and that desiccation destroys the contagium.

The recent researches which have been made on this disease had as a starting-point the important work of Perroncito, an author who was the first to describe the specific bacillus. Toussaint has cultivated the latter in neutral urine, and has declared it to be identical with the ordinary microbes of septicæmia; this opinion, however, is weakened by the negative results obtained by the inoculation of putrid matter. Pasteur has cultivated the bacillus in sterilized chicken-broth; in 1880, after having recognized that the birds which resist the disease possess immunity, he discovered an inoculating process. It was Pasteur who first recommended preventive inoculations for chicken cholera; it was the study of this disease that furnished him the first elements of the method of inoculation. Among the other bacteriological researches we must particularly mention those of Kitt.

Bacteriology. The agent of chicken cholera is an ovoid bacteria, which is extremely small and of the length of 0.3μ to 1μ . It appears in the form of a very short stick, which is contracted in its median part, and consequently takes the shape of a biscuit or of the figure 8; these characters can only be seen with a high-power lens (immersion); with slight enlargement we observe only extremely tenuous points.¹ Cultures in gelatin give small white hyalin islands, which occur in groups and may attain the dimensions of a pin's head (colonies); upon their surface they form by their confluence a thin layer of a dull white coloration. The

¹ The bacillus of chicken cholera is refringent in its central part (bacillus with clear space), aërobic and mobile; it is easily colored with aniline dyes, but it takes neither Gram's nor Weigert's stains.—N. D. T.

microbe is easily cultivated in neutralized chicken broth, with an addition of gelatin or agar; it swarms also in peptonized broth, in a sugar solution of meat extract, in the albumin of a cooked egg, and in the serum of coagulated blood. The most favorable temperature at which this germ vegetates is between $+30^{\circ}$ and $+40^{\circ}$ C. The disease may be transmitted to the gallinaceæ, rabbit, and white mouse, by cutaneous and subcutaneous inoculation, and by the ingestion of blood, excrements, and virulent cadaveric remains. The infected subjects perish in from twelve to forty hours after inoculation. In the guinea-pig, at the point of inoculation abscesses develop in which we find numerous bacilli (Pasteur); we observe also local suppuration in the sheep, the horse (Kitt), and even in man, when the virus is deposited upon skin wounds (Marchiafava et Celli). When injected into the udder of the cow, the bacilli produce a catarrhal mastitis, and are preserved in it for a long time (Kitt). The dog and cat are not contaminated by the ingestion of cadavers. The meat of diseased subjects is harmless for man when eaten. Zürn, however, has mentioned a case of infection due to that cause.

The bacillus of chicken cholera belongs to the category of slightly resistant virus. Desiccation, the majority of disinfectants, diluted sulphuric acid, hydrochloric acid ($\frac{1}{5}$ per cent.), and boiling water destroy it. It is killed by a temperature of 45° to 50° C., prolonged for three-quarters of an hour; a very low temperature (-14° C.) acting for a much longer time (fourteen hours) has no influence on it (Kitt). When associated with other microbes it preserves its virulence for three months. According to Kuppe and Kitt, the pathogenic agent of chicken cholera is identical with that of septicemia of the rabbit.

AFFECTED ANIMALS AND MODES OF PROPAGATION OF THE DISEASE. Cholera is observed in all domestic poultry—chicken, goose, duck, pigeon, turkey, peacock, pheasant; and in all pet birds—parrot, canary, etc.; it appears to exist also in some species living at liberty—sparrow, crow, chaffinch, etc. It frequently makes enormous inroads in poultry yards.

The infection is generally produced by ingestion with food or drink of excrementitious matter which has been ejected by the patients; it is also possible that in some isolated cases it is produced by cutaneous wounds. The remains of cadavers or of slaughtered animals are quite often the causal factors. Cholera is

ordinarily imported into chicken yards by newly-purchased subjects, by poultry belonging to the neighborhood, and by pigeons from a strange pigeon coop where it exists. According to Barthelémy, the infection may even be carried through the intermediation of eggs from diseased chickens; in the affected organism the bacillus is supposed to invade the egg, as it does the foetus of the mammifer (guinea-pig).

Rüchner has demonstrated experimentally, by the method of inhalations, that the bacilli may force their way through the healthy pulmonary muscular mucous membrane, and thus produce infection.

Symptoms. The period of incubation of chicken cholera is very short and its course very rapid. In the greater number of cases death occurs suddenly. In the morning, on opening the aviary, we find several cadavers; we may see birds drop suddenly from their perches; breeding hens often die sitting on their eggs.

The duration of the trouble is sometimes but a few hours. The patients stop eating and keep away from the healthy birds; they are depressed and weak; the feathers are erect and the wings drooping; the crop is distended and the cervical stem is bent. The temperature is increased from 1° to 3° C. (Salle, in 1853, observed this hyperthermia.) In some animals we see a frothy buccal discharge and vomiting; diarrhea soon appears; the fecal matters are at first doughy and yellowish-white; later, mucous, and then serous, of green color and fetid odor; they soil the circumference of the cloaca; thirst is intense. Respiration is laborious; we perceive particular wheezing bruits and râles; there is often hiccough. Gradually the comb becomes dark red or bluish; weakness increases; the birds stagger, fall, and make useless efforts to rise; then death takes place at times in a state of deep drowsiness, in other instances preceded by trembling and convulsions.

Exceptionally, but especially when the disease is prolonged in a chicken yard, its course may be less acute, and its duration reach or even exceed a week. In experiments in transmitting cholera by the digestive canals, Semmer has seen death occur within eight to twenty-one days.

Pathological anatomy. The principal alterations are observed in the intestine, heart, and lungs.

1. The intestine is red externally; its mucous membrane, espe-

cially in the intestinal grêle, is of a uniform dark red coloration or strewn with bloody spots. The epithelium is often deficient, especially on the surface of the papilla; croupous neoproductions and ulceration of follicles are not rare. The mucous membrane of the large intestine, rectum, jabot, and œsophagus is often colored red. The intestinal contents are at times very liquid and frothy, at other times muco-purulent and of a yellowish color, or bloody or chocolate-colored.

2. The heart is dotted with ecchymoses (subepicardiac hemorrhages), the pericardium contains a small quantity of serous liquid; sometimes we observe the alterations of myocarditis and pericarditis.

3. The lungs are congested, reddish-brown, or hepatized (croupous and hemorrhagic pneumonia); they are sometimes covered with fibrinous clots. We may also find laryngitis, tracheitis, catarrhal bronchitis, and pulmonary œdema. These lesions of the respiratory apparatus are usually observed in land birds; aquatic species more frequently show intestinal and cardiac alterations.

Finally, in the blood and in all the organs, the specific bacilli are found in large numbers.

The other lesions present nothing characteristic. The meat has ordinarily a normal aspect, especially when the disease has rapidly developed; in some cases it is of a dark reddish-gray color and undergoing fatty or lardaceous degeneration. On the skin cadaveric bluish-black spots may appear very rapidly.

In cases which run a chronic course there sometimes exist in the lungs and intestines caseous centres similar to those of infectious pneumonia of the pig, and in the centre of which the pathogenic elements swarm (Sticker).

Diagnosis. The *intra-vitam* diagnosis is based on the [epiornitic—W. L. Z.] nature of the trouble, its subacute course, and on the characters of the diarrhea. At the autopsy we find lesions of the intestines, heart, and lungs; a microscopical examination permits us to define the trouble with absolute certainty. We cannot confound the disease with diphtheric inflammation of the mucous membranes; this latter evolves much more slowly, and in most cases it is distinctly characterized by a diphtheric exudate of the buccal mucous membrane.

At times we find some difficulty in differentiating cholera from acute poisoning. The owners in such cases frequently believe

in poisoning. The microscopic examinations establish the diagnosis.¹

Under the name epizootic [epiornitic—w. l. z.] dysentery Lucet has described an infectious disease peculiar to chickens and turkeys; it is characterized by depression, a downcast condition, complete loss of appetite, intense thirst, and an abundant diarrhea, which is at first mucous and bluish-green, then yellowish, and lastly reddish and bloody. The temperature gradually falls from 2° to 3°.

Kitt calls attention to the fact that the diagnosis may be made clear by the local alterations which take place at the inoculating point in birds which are infected artificially. In the pigeon there invariably develops a hard, yellowish mass; if the skin which covers it is removed, we find on the surface of the muscles over an area nearly equal to that of a 10-cent piece a straw-colored exudate, which is dry and hard, and on section shows also a yellowish coloration. In the chicken, at the inoculating point, the muscular tissue is tumefied, indurated; it becomes lardaceous and shows a white coloration.

Treatment. The very rapid course and extreme gravity of the disease (mortality from 90 to 100 per cent.) do not permit of any efficient intervention by means of therapeutic agents. Authors have recommended internally sulphate of iron in solution of $\frac{1}{2}$ to 1 per cent., much diluted hydrochloric acid and tannin in a solution of 1.5 to 2 per cent. Nocard has several times arrested the infection by subcutaneous injections of 5 per cent. phenicated water.

The prophylaxis is much more important than the curative treatment. It is indicated from the first to separate the healthy subjects and to cleanse the coop carefully. The soil, walls, perches, and the utensils used in the poultry yard must be thoroughly disinfected with boiling water, lye, or, better, with a 1 : 1000 sublimate solution, or 5 per cent. phenic acid. The entire

¹ Nocard has given the name of *sleeping disease* to an affection of the chicken determined by a small-sized bacillus, which is especially numerous in the spleen. This disease, which was at first taken for a form of cholera, is particularly characterized by torpidity and almost continuous sleep. The affected animals die within eight to fifteen days. Death, which is quite a frequent ending, generally occurs from the ninth to the thirteenth day. This disease is determined by an anaërobic and aërobic bacteridium, which is easily cultivated in peptonized calf-broth. It is differentiated from chicken cholera by its much slower course, its relatively slight contagiousness, and its non-inoculability in the rabbit. (See Pasteur: *Annal. de l'Institut.*, 1891, and *Receuil Vét.*, *Ibid.*)—N. D. T.

stable should be disinfected by chlorine or sulphur vapor, and the walls whitewashed; the cadavers and excrements must be burned or deeply buried. Disinfection should be repeated several times.

Inoculation. The inoculation of chickens with bacilli of attenuated virulence has been advised by Pasteur as the best means of combating cholera. This author has noticed that weakened, attenuated virus leads only to local tumefaction on the surface of the inoculating point, while strong virus produces necrosis (without suppuration) of the subjacent muscles. The inoculated birds undergo disturbances which they resist, and acquire immunity. Pasteur has observed that some chickens become entirely refractory only after two or three inoculations. He has therefore studied a special mode of inoculation, by which we first inject a much-weakened virus (first virus), and later inject a stronger virus (second virus). A diminution of the virulence is obtained by exposure of the cultures to atmospheric air, prolonged for three to ten months. Pasteur ascribes it to the action of oxygeu.

In 1885 Cagny inoculated 63 chickens in an infected chicken-yard. Both inoculations were made within a space of twelve days. After the first all the birds, with the exception of the ducks, became sick—9 died; after the second inoculation the number of deaths was 8. In a second experiment, made on 36 chickens, 8 died after the first and only 2 after the second inoculation. The results were thus very favorable. Cagny explains the cases of death which occurred after and in spite of inoculation by the natural contamination of subjects which were not yet immune.

In the inoculations made by Kitt upon perfectly healthy chickens with virus which was furnished by Pasteur the chickens were killed by the first virus when it was injected into the pectoral muscles; if inoculated at the extremity of the wing, they resisted. The first inoculation also killed the small birds and the pigeon; the second did not produce any striking morbid phenomenon, but the birds gradually emaciated. The inoculation of the virulent virus was fatal to all the inoculated birds. Kitt denies any practical value from the operation; he considers it as even dangerous and capable of propagating the [epiornitic infection—w. L. z.].

In the inoculations which were practised by Hess in Switzerland, in 1886, on 16 infected chickens, the first inoculation did not produce any manifest reaction; after the second, which was made twelve days later, several animals were seriously affected.

CHOLERA OF THE CANARY AND THE DUCK.

According to Cornil and Toupet, we may observe in canaries and ducks special infectious diseases which are different from chicken cholera. The symptoms and anatomical alterations are precisely the same as in the latter disease, and the blood contains ovoid bacteria ; but by inoculation we produce a fatal disease in only the canary and the duck ; the other birds (chicken, pigeon) resist.

MYCOSIS OF THE PARROT.

Under this denomination Wolff has described a new infectious disease of the parrot. For fifteen to twenty years it has existed in gray parrots (*Psittacus erithraceus*, jaco) of recent importation, which it kills by thousands. Contamination is produced by transport ships coming from the western coast of Africa.

Etiology. This disease of parrots is caused by the *Streptococcus pernicius* (Zopf). The development of this microbe is favored by tainted food, bad water, uncleanness of the boxes in which the birds are transported, and the vitiated atmosphere of the hold of the ships.

Symptoms. The principal symptoms are: loss of appetite, depression, drooping wings, diarrhea, sometimes vomiting; the birds which are very weak remain continually in a crouching position; later we observe convulsions. The disease generally ends in death.

Autopsy. The characteristic lesions exist in the liver; more rarely in the lungs, spleen, or kidneys. These organs present sub-miliary, miliary, or more voluminous tubercles, which are hard, gray or whitish-gray, and contain a very large number of pathogenic streptococci, which determine necrosis of the tissues they invade. We find also enteritis and lobular pneumonic centres.

EPIZOOTIC DISEASE OF WILD ANIMALS AND OF OXEN.

AFFECTED ANIMALS. Under the denomination of "*epizootic disease of wild animals and of oxen*" (*Wild und Rinderseuche*), Bollinger described, in 1878, a disease prevalent in the neighbor-

hood of Munich. It was observed in an enzootic state in wild animals of the royal parks, and afterward in the bovine species. The animals primarily affected were the stag, wild boar, and then the ox; later a few cases were seen in the horse and dog. Among the animals in parks it caused 387 deaths: 153 stags or deer and 234 wild boars. It reappeared in the following summers. In 1881 it affected quite a considerable number of domestic animals in Northern Bavaria. It exists also in other countries. In Prussia, in the district of Kassel, it kills yearly about 100 oxen; its mortality was particularly low (11 cases only) during the years 1885 and 1886.

This disease is not so recent as might be believed. In comparing its symptoms with those described in certain old monographs, we acquire the conviction that it was formerly known. The terrible epizootic described in 1858 in the *Veterinarian*, an epizootic which decimated the bovines and wild ruminants, was nothing else than the disease in question. It has generally been likened to bacterian anthrax. It presents a marked symptomatic analogy to "gloss-anthrax," and it has been asked if part of the clinical facts reported under this title are not related to this disease.

Etiology and pathology. At the present time the infectious agent of the epizootic disease of wild animals and of oxen is yet unknown. According to Friedberger and Hahn, while the horse succumbs as a consequence of the inoculation of infectious matter taken from the ox, animals of the bovine species resist; with these the effects of the virus are limited to a local tumefaction. In the serum of œdema Friedberger and Franck have found micrococci and very small bacilli. Bollinger has successfully inoculated this disease in the horse, pig, sheep, goat, and rabbit. Animals to which he administered a certain quantity of bloody intestinal contents found in subjects affected by the exanthematous form succumbed to the pectoral form within fifty-four hours; and, reciprocally, by inoculating the blood and pleural exudate of individuals which have died from the pectoral fever, he has produced the exanthematous form. He has therefore furnished a proof of the identity of these two clinical varieties and of the relationship of the process.

No case has yet been mentioned of contagion to man. Accidental wounds (operation, autopsy) have never had any unfavorable consequences, and the meat of animals that have been killed has always been consumed with impunity.

Infection seems to be possible by the skin (exanthematous form) as well as by the pulmonary mucous membranes (pectoral form) and intestine (intestinal form). The experiments of Bollinger have established that the disease may assume the pectoral form when the infection takes place through the intestinal canal. The wild boar is very probably contaminated by the ingestion of the remains of infected animals. The exanthematous form appears to have as a principal cause the penetration of infectious matter by small cutaneous wounds (fly and mosquito stings, wounds of the head, of the buccal mucous membrane, etc.). Direct contagion (from animal to animal) has never been observed, but the epizooty has been propagated by the sale of meats and skins in localities which were previously free from it. Finally, Zeidler reports that an owner, after having found and handled cadavers of wild animals, communicated the disease to subjects of his own stable.

In the blood and cadaveric remains of oxen, horses, pigs, etc., dead of an unknown epizootic disease, which existed in Simbach in 1885, Kitt found short thick bacilli of the length of 0.6μ , and 0.3μ wide, which were markedly colored toward the extremities; they were particularly abundant in the blood. By inoculation he recognized that these microbes are pathogenic for the mouse, rabbit, pigeon, and caged birds; also for the pig, goat, horse, dog, sheep, ox; in the rabbit they invariably produce a hemorrhagic tracheitis; they resemble greatly those of septicemia of the rabbit, of chicken cholera, and infectious pneumonia of the pig. Kitt admits the identity of this disease with the epizootic disease of wild animals, especially on account of their transmissibility to the same animal species and the similarity of the anatomic pathological alterations which they determine.

In blood preparations from animals which had died of the epizootic disease of wild animals, and which had been preserved since 1878, he found bacilli similar to those which have just been described. From specimens which Kitt sent to him, Hüppe confirmed the statements of this author, and reached the same conclusions. He admits, moreover, that the epizootic disease of wild animals may appear in the form of a simple septicemia, intestinal mycosis, or infectious pleuro-pneumonia; he proposes to give it the name "hemorrhagic septicemia." He has inoculated rabbits, guinea-pigs, and mice with cultures of bacteria of infectious pneumonia of the pig, of the infectious disease of Kitt, of chicken cholera

and septicemia of the rabbit; all these diseases produced in the animals experimented upon similar alterations; their pathogenic agents behaved in the same manner in the tissues and in the blood. He considers these morbid conditions as identical with contagious pneumonia of the horse; he describes them as special forms of "hemorrhagic septicemia."

Pathological anatomy. The anatomical alterations of the epizootic disease of wild animals permits us to recognize therein the *exanthematous*, *pectoral*, and *intestinal* forms. The first two are the most important; the third is frequently but a complication of these. Let us add that in a large number of cases they are found associated.

1. The *exanthematous* form is marked at first by an enormous tumefaction of the skin and subcutaneous connective tissue. The integument of the diseased regions (head, neck, shoulders, etc.) is inflamed and oedematous. The subcutaneous connective tissue is the seat of a sero-gelatinous infiltration; we observe sometimes a limpid serum, varying from a bituminous to a golden-yellow color, and sometimes extensive hemorrhagic centres. The lymphatic ganglions of the neighborhood (submaxillary ganglions, superior cervicals, etc.) are infiltrated with serum and blood and considerably hypertrophied.

The buccal mucous membrane and the submucous connective tissue may present similar alterations. The first is cyanosed, thickened, and infiltrated. The tongue, which is often considerably hypertrophied, is wrinkled on its lateral fascia. In a case observed in the ox by Friedberger it had a thickness of ten centimetres and weighed four kilogrammes; its color was of a dirty red-brown; deep bloody suffusions were found in it, which were of the size of a saucer, and covered only by the epithelium; its tissue was moist and infiltrated; the sections showed a purple color, from which escaped a plasmatic yellowish liquid, which coagulated rapidly. The pharyngeal mucous membrane presented similar alterations. The salivary glands were anemic and dried.

The respiratory mucous membrane is often marked by similar alterations. In the larynx, trachea, and bronchi it forms quivering wrinkles which reduce the diameter of these ducts considerably. Sometimes we find in the bronchial canals croupous lesions.

Hemorrhages exist in all the organs, especially in the serous membranes, muscles, and lungs. The spleen is normal; its pulp is

only a little less moist than usual; the blood has also its ordinary properties and color. The perineal connective tissue is infiltrated with blood. The hemorrhagic enteritis is nearly a constant lesion.

2. In the *pectoral* form the lung is hepatized (croupous pneumonia) and the interstitial connective tissue is the seat of a gelatinous infiltration. The pleura is inflamed, tumefied, and covered with a sero-fibrinous or plastic exudate (serous or sero-fibrinous pleurisy); in the pleural cavity we find a variable quantity of liquid (as much as 30 litres). We observe also an inflammation of the mediastinum and pericardium. This form is also accompanied by hemorrhages which are disseminated in the whole organism; we also frequently recognize a hemorrhagic enteritis. The blood and spleen appear normal.

3. In the *intestinal* form the digestive mucous membrane, especially that of the intestinal grêle, is tumefied and studded with hemorrhages of greater or less extent (hemorrhagic enteritis); the epithelium is desquamated, the intestinal contents are liquid and bloody. With these alterations we ordinarily find but slightly accentuated those of the exanthematous and pectoral forms.

In subacute cases these various lesions are but little marked.

Symptoms. The *exanthematous* form is the most common in cattle. It is indicated by disturbances of appetite and lacteal secretion. The temperature ranges between 40° and 42° C. The soft parts of the head, face, fetlock, neck, and shoulders are often the seat of a considerable, distended, hard, hot, and painful tumefaction, which is at times œdematous and preserves the imprint of the finger. The invaded regions are deformed; the tumefaction frequently reaches the thickness of 20 centimetres.

We observe, besides, symptoms of stomatitis and pharyngitis. The animals are affected by dysphagia; sometimes the jaws execute continual convulsive movements. Long threads of saliva escape from the mouth; the tongue is frequently protruding, tumefied, and twice or three times its normal size. It shows a reddish-blue color and depressions are produced by the teeth. Upon its lateral fascia the mucous membrane forms wrinkles which are of slight consistency and quivering. The enormous tumefaction of the buccal and pharyngeal folds may produce attacks of suffocation and lead to asphyxia. The other mucous membranes of the head are colored reddish-brown and are studded with hemorrhages, the vaginal mucous membrane, also, is sometimes of an intense red.

Toward the end of the disease a much-marked dyspnœa and colics occur; the animals complain, lie down frequently, make violent expulsive efforts, and eject softened diarrhœal excrementitious matters, which are mixed with cronpous products. Weakness increases; the decubitus is soon permanent. Death occurs within twelve to thirty-six hours (minimum, six hours; maximum, three to four days).

The *pectoral* form is generally found in wild animals. It is very rare in subjects of the bovine species; we possess, therefore, but very incomplete facts regarding its symptomatology. In the ox, in exceptional cases where it has been observed, it was marked by symptoms of pneumonia and intense dyspnœa. Its duration, which is longer than that of the exanthematous form, varies from five to eight days.

In the ox the *prognosis* is very serious. According to Bollinger, the mortality exceeds that of anthrax. In 95 animals observed by Putscher, 9 recovered (mortality, 90 per cent.).

Differential diagnosis. This disease has been confounded with anthrax and plenro-pneumonia.

1. It resembles *anthrax* in the pseudo-carbunculous tumefactions of the skin and buccal mucous membrane, the hemorrhagic enteritis, and the hemorrhages which occur in the organs. Let us remark that anthrax frequently exists in an epizootic state in species confined in parks. The epizootic anthrax, which existed in the "Grünwald," near Berlin, in 1874, destroyed 2000 animals (Bollinger). Notwithstanding these common symptoms, this disease is distinctly differentiated from anthrax by the following characters:

a. By the absence of the bacteridium, which has not been found in any of the numerous cases studied from a bacteriological standpoint.

b. By the absence of tumefaction of the spleen and the turbid consistency of the blood, which lesions are constant in anthrax.

c. By its very easy transmission to the pig, an animal which possesses almost entire immunity in regard to anthrax.

d. By its difficult transmission to the sheep, which is so very sensitive to the anthrax virus.

e. By harmlessness of the diseased meat for human beings. Man may consume it without danger, and appears to possess complete immunity.

The doubts which have arisen of late as to the independent existence of these diseases are therefore not justified.

2. In the beginning of these epizootics we may confound this disease of oxen with *pleuro-pneumonia*; but the autopsy furnishes precise information. In the former the pulmonary lesions are uniform and contemporary; we know that this is not the case in *pleuro-pneumonia*, the course of which is also much slower. Here a diagnostic error might have serious consequences; if prophylactic inoculations were practised, they would be followed by serious losses.

3. It may also simulate *malignant œdema*.¹ But while it is transmissible by the digestive organs and by cutaneous inoculation, malignant œdema can only be communicated by subcutaneous inoculation. This remark applies also to symptomatic anthrax, which is sufficiently characterized by its localization and by the crepitation of the tumors which it produces.

Treatment. We do not know of any efficient treatment. The subcutaneous injection of a 1 per cent. alcoholic solution of phenic acid and the administration of salicylic acid, which were tried by Friedberger, have not given any satisfactory results. We might perhaps, as for symptomatic anthrax, make large incisions in the tumefied region, and use antiseptic measures. From a sanitary standpoint the disease requires the same treatment as bacterian anthrax.

BARBONE OF THE BUFFALO.

In Italy, under the name of *barbone*, is designated a disease of the buffalo which is quite frequent and well known in the neighborhood of Salerno, Rome, and Terra di Lavarò. *Barbone* presents a great similarity to epizootic disease of wild animals. I was described for the first time in 1816 by Metaxa; at more recent dates it has been likened to typhus, anthrax, strangles, etc. The researches of Oreste and Armauni² have cleared up the nature of this disease.

Barbone is observed in summer and appears in young animals. It begins with intense fever (41°–42° C.), depression, and loss of

¹ Malignant œdema is *extremely rare* in the ox, and only isolated cases are observed.

—N. D. T.

² Oreste and Armauni: *Studie e Ricerche al Barbone dei Bufali*, Napoli, 1886; *Sull' Attenuazione del Virus del Barbone*, Napoli, 1887.

appetite. Among its principal symptoms we may mention a tumefaction of the intermaxillary space, ptyalism, discharges, inflammation of the buccal mucous membrane and of the tongue, and intense dyspnœa. Tumefactions may appear on the abdomen, neck and shoulders, face and legs; they are œdematous, preserve the imprint of the finger, and do not crepitate. On section they are found to be composed of a yellow gelatiniform exudate. In sub-acute cases the animals are stretched upon the ground and die in convulsions in from ten to twenty-four hours; but death may occur much more rapidly (in three to six hours). When the disease is prolonged longer than twenty-four hours a cure is possible. In a determined region the average duration of the epizooty is from nine to ten days. The mortality amounts to 40 or 50 per cent.

At the autopsy in the tumefied regions and subjacent muscles we find the connective tissue infiltrated with a yellowish gelatiniform exudate. The spleen is normal. The blood contains ovoid bacteria which are similar to those of infectious pneumonia of the pig, chicken cholera, and septicemia of the rabbit. They are found in the exudates, blood, saliva, urine, milk, and in pregnant females in the foetal blood. *Barbone* is transmissible to the horse, ox, sheep, pig and guinea-pig, to the rabbit, pigeon, and turkey. The dog is refractory to it. Infection naturally takes place through the skin (wounds), through the digestive mucous membrane, more rarely through the pulmonary tissue (modes of infection which are similar to those of epizootic disease of wild animals). The infectious agent is destroyed by desiccation, boiling water, 2 per cent. solution of phenic acid, and 5 per cent. solution of sulphuric acid.

Oreste and Armanni have recommended inoculation as a prophylactic means. They have succeeded in weakening the virus by inoculating it in the pigeon. Three inoculations made at a few days' interval with a small quantity of blood taken from infected pigeons sufficed to give the buffalo immunity.

SYMPTOMATIC ANTHRAX.

HISTORY. It is known under the names of *black gangrene*, *flying disease*, *flying gangrene*, *cold gangrene*, *disease of the thigh*, *of the legs*, *carbunculous disease*, and *anthrax emphysema*. Symptomatic anthrax was described a short time ago as a form of anthrax

fever. For a long time it was known to be *non-contagious*, and that the meat of diseased subjects may be consumed with impunity by man and animals. Some old authors have written somewhat exact monographs of it. Wallraff in 1856 has very well described the symptoms of emphysematous anthrax by proving the insensibility of the tumors at the time of incision. In 1870 Pfisterer was able to convince himself that this disease differs in its nature from anthrax fever. However, in order to find a definite and satisfactory theory concerning the fact that symptomatic anthrax and anthrax fever constitute two morbid entities which are essentially different, we must make use of the etiological researches of Feser and Bollinger.

In 1860 Feser discovered very fine mobile bodies in the muscular serum of animals which had died of symptomatic anthrax; in 1875, in pursuing his researches on bacteridian anthrax in the Alps of Northern Bavaria, he made at the same time a thorough study of emphysematous anthrax. He considered these very small club-shaped bodies which were animated by undulatory movements as the pathogenic agents of the disease. In the ox, sheep, and rabbit he produced symptoms characteristic of the affection by inoculating, under the skin, earth from infected regions. About the same time Bollinger also discovered the bacillus of symptomatic anthrax; he describes it as a very fine rod, which is elongated and possesses rotary movements; he produced the disease experimentally in the ox, sheep, and goat by subcutaneous inoculation of the anthrax blood.

The principal modern researches on the nature of symptomatic anthrax are due to Arloing, Cornevin, and Thomas. These authors have well described the bacillus; they have studied its biology, and have finally discovered a process of inoculation.

AFFECTED ANIMALS. Symptomatic anthrax is an enzootic infectious disease of the ox, which is stationary in certain countries. In stables and pastures in some regions (especially in the Alps) this disease exists the whole year, but particularly during the warm season (summer and fall); it seems to have a preference for localities with swampy soil.

In Germany the principal districts with symptomatic anthrax are the Alps of upper Bavaria, certain districts of Schleswig-Holstein (Apenrade, Hadersleben, Husum, Steinburg, Norderdithmarschen, Süderdithmarschen, Toudern, and of the Rhine province

(Wiesbaden, Cologne, Treves, and Düsseldorf). It is also very frequently seen in Würtemberg and Baden, more rarely in Saxony. In Switzerland it is especially frequent in the cantons of Berne, Graubünden, Glarus, Triburg, Unterwalden, and Schwyz. In Austria it exists in the Alps of Vovarilberg (forest of Bregenz), Salsburg, Tyrol, Carinthie, and Stiermark, in the districts of Scheibbs, Lilienfeld, and in the valley of the Ems. In France it is found in numerous departments (Haute-Marne, Cantal, Puy-de-Dôme, Hautes-Alpes, Basses-Alpes, etc.). It is also seen in Belgium, Italy Algiers, and a few other countries.

In general it affects animals that are from six months to four years old. Calves which are less than six months do not contract it; as long as the lacteal diet lasts they are not exposed to the infection of pastures. Let us add that the calf is much less sensitive to experimental inoculation than adult animals; it withstands a dose of virus which is fatal for the latter. Subjects more than four years old which have been raised in infected districts have generally been affected once before, and have acquired immunity from the disease (Arloing). But when animals coming from localities where symptomatic anthrax does not exist are led into infected pastures they may contract the disease no matter what may be their age. Experience has demonstrated that a first attack confers immunity in all cases. The pig, dog, cat, and rabbit, the black rat, and man seem to possess a natural immunity from the disease. The meat of animals affected with bacterian anthrax may be eaten with impunity by man, also by the dog and pig. In the horse, donkey, and white rat, inoculation produces but a local reaction (tumefaction).

On the contrary, symptomatic anthrax is very easily inoculated in the ox, goat, sheep, and guinea-pig. The goat and sheep may contract it naturally (Arloing, Hess). Many cases of "symptomatic anthrax" seen in the ox were, but malignant œdema(?). Symptomatic anthrax exists quite frequently side by side with malignant anthrax (in the Alps of Northern Bavaria, for instance); therefore they formerly considered the first as a precursor of anthrax fever.

STATISTICS. In Bavaria during the year 1886 symptomatic anthrax affected 88 oxen and 7 sheep, and in 1887 82 oxen. In Prussia the number of affected bovines in 1886 was 94, and in Austria 376. In Switzerland it was 342 for the year 1887. In

the district of Mörs (Rhine province) symptomatic anthrax occasioned great losses in the years following 1880 on account of the freshets which happened at that period. During these years 12 to 13 per cent. of all the young animals died.

Bacteriology. The bacilli of bacterian anthrax are 5μ to 15μ in length (nearly half the diameter of a red corpuscle), and they are 3μ wide. They possess undulatory and rotary movements, which are quite marked, and they are provided at one of their extremities with a very refringent spore, which gives them the aspect of a club. They are very abundant in the altered tissues, in the subcutaneous connective tissue, muscles, bile, and the intestinal contents (Kitt), but they are rare in the blood; therefore, inoculations made with this liquid generally remain sterile. They are found in large numbers in the blood when the autopsies of cadavers are made a long time after death. This bacillus is anaërobic, like the *Vibrio septicus*; it swarms in the tissues without utilizing the oxygen of the blood; its development is accompanied by the formation of gas; its sporulation takes place within the organism (endogenous development); the exogenous development in the soil is very probable, but it is not positively demonstrated. Arloing was the first to cultivate it in chicken broth. Quite recently W. Koch described the characters shown by cultures on gelatin and potato; they form on the surface of these mediums a reticulated membrane, which is hard, whitish-gray, and disposed in very clean folds. He has established that the bacillus vegetates perfectly well at house temperature and that it liquefies the gelatin by the development of gases (anaërobic microbe); he has likened it to the microbe of emphysematous or mephitic gangrene (overwhelming gangrene) of man. Ehlers has pointed out various evolution forms of the bacillus.¹

¹ The microbe of symptomatic anthrax (*Bacterium Chauvæi*) appears under several aspects. It may take the five following forms: 1, as a *nucleated, sporulated bacterium*, which is of uniform size or enlarged at the surface of the spore; 2, as an *articulated bacterium* in its centre, and provided with a spore at each extremity; 3, as a *long homogeneous bacterium*, which possesses great mobility; 4, as a *fusiform bacterium*, with or without sporules; 5, as a *micrococcus* (Arloing, Cornevin, and Thomas). We may find these various types in the anthrax tumor; the fusiform bacteria appear late (from twenty-four to thirty-six hours after death). During life the blood is poor in microbes, but after death we find therein a large quantity of micrococci and mobile bacteria. The *Bacterium Chauvæi*, anaërobic, can only be cultivated *in vacuo* or in inert gases. It takes neither the Gram nor the Weigert tests, but it is easily stained with aniline colors.—N. D. T.

The researches of Arloing, Cornevin, and Thomas have shown that the virus of symptomatic anthrax possesses a great resistance to the various agents of destruction. If dried, it preserves its activity for a long time. In the soil the virulence of the cadavers persists for six months; the bacteria of putrefaction and bacteridium do not exercise any influence on it. Intense cold no longer affects it. The virulence of dried and pulverized meat resists steam when used in spray (100° C.—Kitt); it is weakened, but not destroyed. The fresh virus is killed in twenty minutes by a temperature of 100° C., and in two minutes by boiling water. If dried, a temperature of 110° C. is required to destroy it, and this must be prolonged for six hours, or the action of boiling water must last for two hours; it is also rendered inactive by vapors of bromine, watery solutions of sublimate (1 per 5000), salicylic acid (1 per 1000), thymol (1 per 800), phenic acid (1 per 50), boric acid (1 per 5), hydrochloric acid (1 per 2), etc. Among the antiseptics which have no influence upon it we may mention: alcoholic solution of phenic acid, quicklime, sulphate of iron, chloride of zinc, and sulphuric acid. These researches have also demonstrated that the virus is attenuated by the prolonged action of high temperature and certain chemical substances. The activity of the weakened bacillus may be increased by the addition of lactic acid (lactate of potash), acetic acid, diluted alcohol (Nocard and Roux).¹

¹ Arloing, Cornevin, and Thomas have established that the attenuated virus of symptomatic anthrax, when added to a small quantity of lactic acid, produces the same effects as ordinary virus. But in reality it does not produce a revivification of the contagium; the *Bacterium Chauvai* does not regain its former activity. Nocard and Roux have in fact demonstrated that lactic acid acts by altering the muscles in which it is introduced and diminishes their vitality; they have obtained the same result by using other substances, or even by a simple contusion of the region where the attenuated virus is injected.

Roger, after having observed that the *micrococcus prodigiosus* favors infection by producing a general modification of the organism, has made numerous experiments upon symptomatic anthrax, which have been mentioned in a work from which we will transcribe the principal conclusions:

1. Natural immunity of animals (rabbits or pigeons) toward symptomatic anthrax may easily be overcome when we associate the agents of this disease with another microbe, whether this auxiliary microbe be a simple saprophyte (*Bacillus prodigiosus*) or a pathogenic bacterium which cannot be harmful in the doses which are used (*Staphylococcus aureus*, *Proteus vulgaris*).

2. The action of these microbes is due to the substances which they secrete. For the *B. prodigiosus*, the active substance is soluble in glycerin and insoluble in alcohol.

3. The active substance acts, not by producing a lesion, as is done by lactic acid, but by determining a general modification of the organism; its influence is specially

Pathology. Symptomatic anthrax is a true infectious disease of wounds; the bacillus invades the organism only through wounds of the skin or mucous membranes, and then these wounds must penetrate as far as the subcutaneous connective tissue (similar to the bacillus of malignant œdema). Inoculations practised in the substance of the derma remain sterile. It is generally admitted that the disease is developed through wounds which the animals may happen to receive when on pasture (on the legs and in the mouth), and that the traumatisms are infected by earth that contains virus. In animals affected by symptomatic anthrax Hess has often found wounds upon the pasterns and cannon. He thinks that infection is easily produced, especially at the time of dental eruption, through the unprotected buccal mucous membrane. According to Hafner, the buccal and pharyngeal mucous membranes are very favorable for the introduction of the infectious elements, especially in subjects kept continually in the stable for some length of time.¹ The result of the observations gathered during the experiments of inoculation is that the period of incubation averages two days

marked when it is directly introduced into the circulatory system; the extract of one drop of culture is sufficient to destroy the immunity of the rabbit.

4. The immunity of the rabbit may be increased by means of intravenous inoculations of symptomatic anthrax; the animal then resists the effects of microbial associations.

5. Inoculation of symptomatic anthrax in the posterior chamber of the eye, in the rabbit, determines a fatal lesion; this lesion permits the development of virus, which is inoculated in other points.

6. The bacillus of symptomatic anthrax secretes matters which seem to have two opposite effects: if introduced at the same time as the virus, they favor its development; if they are injected a few day before the inoculation, they render the animal refractory. This apparent anomaly is easy to explain: the microbial substances which favor the infection act by arresting for a time diapedesis (Bouchard); within a few hours their effect is dissipated and the morbid tendency has disappeared; on the contrary, artificial immunity results from a nutritive modification which requires a certain time in order to be produced, and which is marked by a change in the constitution of vitiated fluid and tissues.—N. D. T.

¹ Symptomatic anthrax may be produced experimentally by introducing the virus into the subcutaneous or intramuscular connective tissue, in the blood, and the digestive canal (Arloing, Cornevin, and Thomas). But natural inoculation takes place, especially by cutaneous wounds, which extend to the subjacent connective tissue. If introduced directly into the circulatory system, the virus of symptomatic anthrax behaves like that of septicemia. The bacteria may swarm therein; but if the vascular endothelium is intact, they are attacked and destroyed by the white corpuscles (phagocytosis). If, on the contrary, they penetrate into the peri-vascular tissues (lacerations, infarcts), they produce one or several anthrax tumors, and, in general, a fatal process, by a mechanism identical with that of the *Vibrio septicus*, which is introduced into the bloodvessels and deposited in a traumatic centre by the blood, bistournage.—N. D. T.

(minimum one day, maximum three to five days). The researches of Arloing, Cornevin, and Thomas have established the possibility of foetal infection through the placenta. When the fruit of conception survives it seems to possess immunity.

Symptoms. Symptomatic anthrax is characterized by subcutaneous crepitant tumors, by secondary tumefaction of the corresponding ganglion, and by locomotor troubles. It is a pyretic disease which almost always ends in death within a very short time (thirty-six hours to three days).

1. The anthrax tumor may appear in different regions: thigh, neck and shoulders, chest, lumbar region, and hind quarters; it is never located above the knee or the calf of the leg. In the beginning it is small and sensitive, but extends very rapidly, and may acquire a considerable size in a few hours, and even invade the whole surface of the trunk. On palpation we recognize therein a crepitant bruit; percussion produces a tympanitic sound. In its central part this tumor is insensitive, dry, and parchment-like; sometimes the skin is gangrenous and cold; incisions made in it are painless and give vent to a dark-red, frothy liquid, which has a bad odor. In some cases there exists but one tumor; in others we find several, which may become confluent. The neighboring lymphatic ganglions, which are greatly tumefied, give to the hand the sensation of neoplasms more or less voluminous.

2. Among the general symptoms we must mention: an abrupt cessation of appetite and rumination, depression, weakness, intense fever (as high as 42° C.), and interference with walking—lameness, paresis of a leg, stiffness—which is due to the development of one or several tumors in the locomotor apparatus. In proportion to the extent of the local lesions, the other symptoms become more marked, the respiration is accelerated, dyspnoeic, and painful; sometimes, also, we notice violent colics. Weakness increases, the temperature becomes lower, and nearly all the animals die within the time mentioned above.

The course of the disease is variable: sometimes we observe first the appearance of anthrax tumors, at other times general symptoms. In old animals we see at times benign cases, in which the local manifestations are insignificant and the fever moderate; then the cure generally occurs within twenty-four hours. Arloing has described another very mild form, which is indicated by digestive troubles (loss of appetite, slight colics, meteorization, weakness, etc.).

Pathological anatomy. The skin covering the lesions of anthrax is mortified (dry gangrene). The subcutaneous connective tissue is infiltrated with blood and serum, and is distended by gases which escape when the tumors are incised. The muscles, which are dirty brown, black, red, or dark yellow, are friable, porous, infiltrated with liquid, and crepitate on incision.¹ On compression turbid blood oozes containing gas having a bad and disagreeable odor. The muscular fibres present various degenerative alterations. The gases of the anthrax tumor are combustible, and burn with a bluish flame. According to Bollinger, they should be inodorous and composed of carburet of hydrogen; carbonic acid has, however, been found there (Arloing). A complete chemical analysis has not been made. Some authors have found them fetid; it is probable, therefore, that sulphuretted hydrogen enters into their composition.² The lymphatic ganglions which correspond to the tumefied regions are obstructed, congested, and studded with hemorrhages and infiltrated with bloody serum. The afferent lymphatic vessels are sometimes distended by gases, and present a moniliform aspect. When anthrax is localized to the bucco-pharyngeal membrane the muscular layers of the tongue and pharynx show alterations similar to those of the muscles of the locomotor apparatus.

The abdominal cavity frequently contains a red transudate or a small quantity of serous liquid; sometimes it is free from alterations; these differences are due to the seat of the tumefactions, as to whether or not the process has invaded the peritoneum. In the epiploon, the mesentery, and in the neighborhood of the kidneys we frequently observe yellowish infiltrations mixed with blood. The gastro-intestinal mucous membrane is tumefied, red, and hemorrhagic; the intestinal contents are bloody; the liver is hyperemic; the spleen is not affected.

In cases where tumors exist in the thoracic walls the pectoral cavity contains a sero-bloody exudate, the pleura and mediastinum are infiltrated; we find hemorrhages in the lungs, the pericardium,

¹ The color of anthrax tumors becomes lighter toward the periphery; in contact with air it may become rutilant (shiny), like that of venous blood when exposed to the atmosphere.—N. D. T.

² The gases which escape from incised anthrax tumors shortly after death have an odor which is not disagreeable. They contain neither oxygen nor oxide of carbon. During the later period of life the blood contains, besides oxygen, nitrogen, and carbonic acid, gases which are non-absorbable by potassium and pyrogallie acid, and which probably have the same origin as those of the tumors (Arloing, Cornevin, and Thomas).—N. D. T.

myocardium, and under the endocardium; the myocardium is very friable; sometimes the bronchial mucous membrane is congested and hemorrhagic.

The blood, which is of normal color, is coagulated. Outside of the affected regions the muscles are little altered; the meat serum has an acid reaction (Feser). The tissues rapidly decompose. The cadavers are greatly inflated by the gas which accumulates under the skin.

Differential diagnosis. It is especially important to distinguish symptomatic anthrax from bacteridian anthrax. We have the following basis as a guide:

1. Clinically symptomatic anthrax is characterized by crepitant, emphysematous tumors (which are never observed in anthrax fever), and also by its non-contagiousness and its extreme gravity.

2. Anatomically it has as principal characters the muscular lesions (emphysema), the normal consistency of the splenic parenchyma and of the blood; besides, the latter is partially coagulated, a character which is never seen in bacteridian anthrax.

3. Bacteriology has defined the morphologic differences of both infectious agents. The bacillus of symptomatic anthrax is short, thick, and rounded at the extremities, one of which is club-shaped and sporulated; it is also animated by very quick movements. The bacteridium is longer, thinner, and of equal thickness; it is distinctly truncated at the extremities; it is immobile and gives characteristic cultures. (See Bacteridian Anthrax.¹)

4. Inoculations of symptomatic anthrax practised on the skin are sterile, while the introduction of a small quantity of bacteridium in the thickness of the derma gives positive results. If it is inoculated into the subcutaneous connective tissue, symptomatic anthrax produces enormous tumefaction; anthrax fever determines a swelling much less marked. The intravenous injection of the virus of symptomatic anthrax produces only an insignificant affection and confers immunity; the same operation made with the virus of anthrax fever is fatal. The guinea-pig invariably dies from the inoculation of symptomatic anthrax and anthrax fever.²

¹ The bacterium takes the Gram and Weigert stains; the bacillus of symptomatic anthrax is not colored by these two processes.—N. D. T.

² In order to differentiate easily and surely symptomatic anthrax from anthrax fever, it is sufficient to inoculate at the same time a rabbit and a guinea-pig. If only the latter dies, the disease is symptomatic anthrax; if both animals die, we have bacteridian anthrax.—N. D. T.

while the rabbit (which is extremely sensitive to the latter) and the mouse are naturally refractory to symptomatic anthrax. In general, the animals are less sensitive to symptomatic anthrax than to anthrax fever. (For the differential diagnosis of symptomatic anthrax and septicemia, see Malignant Œdema.)

Treatment. The malignant character of the disease and the rapidity of its course generally render any therapeutic intervention useless. In the beginning we may try disinfection of the subcutaneous connective tissue by means of large incisions made in the skin. When the tumor is located on the lower extremity of a leg Wallroff recommends the application of a ligature above the tumefaction and scarification. We fail to see the usefulness of this operation. The principal prophylactic indication is to avoid infected pastures. In Northern Bavaria woods have been planted in vast territories in order to keep animals from them (Zeilinger). The infection being very rarely produced through the intestinal canal, Kitt has proposed giving the hay grown in infected pastures in the stable; he has also advised not to lead to pasture animals having any wounds on their legs nor those which are shedding their teeth. We may remark, nevertheless, that the disease may originate in animals kept permanently stabled (Baden, Würtemberg).

The sanitary police measures enforced for anthrax fever should be applied to symptomatic anthrax. This is done in some countries (Prussia, Würtemberg, etc.). In Austria special measures concerning bacterian anthrax were prescribed by an edict of April 10, 1885 (obligatory declaration: prohibition of slaughtering the diseased animals and utilizing the cadavers with the exception of the skin; destruction of the cadavers and their remains; disinfection; and periodical visits of the sanitary police).

PREVENTIVE INOCULATION: METHOD OF ARLOING, CORNEVIN, AND THOMAS. Preventive inoculation against symptomatic anthrax has assumed of late years a special scientific and economic importance. Arloing, Cornevin, and Thomas have established that subcutaneous and intramuscular inoculations always lead to death, while an intravenous injection of the virus determines but a few insignificant general symptoms and at the same time confers immunity. Intratracheal inoculation has given similar results. All attempts at inoculation through the gastro-intestinal canal have failed(?). Taking these facts as a basis, the authors inoculated in

1880, at Chaumont, 13 animals of the bovine species by injecting directly into the jugular vein virus diluted with distilled water. The subjects thus protected could be inoculated with impunity six months later with the ordinary virus injected under the skin. Of 12 animals present which had not undergone preventive inoculation, 9 died from symptomatic anthrax and 2 others were seriously sick. Two hundred and forty-five animals were inoculated with the same success in the Department of Haute-Marne in 1881, and 78 in the Department of Ain in 1882. But intravenous inoculation is a very delicate operation: the jugular vein must be laid bare in order to avoid penetration of the virus into the subcutaneous and perivenous connective tissue. Experimenters, however, have abandoned this early method. They have selected for the inoculating point the caudal extremity; in this region the virus produces but a passing tumefaction, while at the same time it confers immunity. Moreover, they operate with a virus which is attenuated by heat. The mild character of the phenomena consecutive to inoculation practised at the end of the tail is explained by the density of the connective tissue in this region, and by the temperature, which is considerably lower than in other parts of the body.

The attempts which have been made of late years in France, Switzerland, Austria, and Germany establish the fact that inoculation against symptomatic anthrax considerably reduces the rate of mortality due to this disease.

The inoculating virus is prepared in the following manner: 40 grammes (1 part) of diseased muscle are dried at a temperature of 32° C., and then well mixed with 80 grammes (2 parts) of water. The mixture is divided into twelve parts of 10 grammes each, which are separately placed upon a flat plate and dried in an oven for six hours. Six plates are to be dried at 100° and the other six at 85°. The contents of the former give a weak virus—the first virus; that of the latter furnishes the second virus. Thus prepared, the virus may be preserved for quite a long time.

In order to practise the first inoculation we take 0.1 gramme of the crust which has been dried at 100° and dissolve this in 5 grammes of water, afterward using a Pravaz syringe (constructed according to Cornevin's indications, by Lépine, instrument-maker, in Lyons, France). The mixture, after being filtered through fine muslin, is injected subcutaneously, $\frac{1}{2}$ cubic centimetre per head. The

piston of the syringe is fitted with a graduated stem, which enables us to measure the doses exactly.

The first inoculation is made upon the inner face of the tail at a distance of three widths of the hand from its free extremity. The hair must be cut beforehand. A fine trocar is then introduced, point upward, to a distance of 8 centimetres between the skin and bones; when the trocar is withdrawn we introduce into the opened track the canula of the syringe, after having first shaken it in order to render the liquid virus equally active throughout the whole mass. The injection is performed by gently pushing the piston. In order to prevent escape of the virus we apply a finger upon the opening made by the trocar, and, by compressing the track of the injection upward, we thus force the liquid into the neighboring connective tissue. When the puncture produces a slight hemorrhage we wait till the bleeding stops or make a new puncture. Finally, in order to prevent the escape of the inoculated matter, we apply to the wound a piece of rubber plaster about two centimetres wide.

Three assistants must hold the animals. We may inoculate twenty to twenty-five subjects per hour. The second inoculation is made ten days after the first; the *modus operandi* is the same; it is performed at a distance of eight inches from the lower extremity of the tail. The end of winter and the spring are the most favorable seasons for this inoculation. It does not produce any appreciable morbid reaction.

STATISTICS OF THE INOCULATION OF SYMPTOMATIC ANTHRAX. In 1883 Cornevin inoculated 125 animals in France. The results were excellent. The inoculations made in 1884 have also given good results.¹

In Switzerland, in 1884, most inoculations were made by Hess and Strebel. The number of inoculated subjects numbered 2000; they were led to the Alpine pastures, where symptomatic anthrax existed. In the inoculated subjects the rate of mortality was 28 times less than in non-inoculated animals (Strebel). In 1885 they

¹ In France during the year 1887 symptomatic anthrax was observed in 45 departments (counties): in 34 there were seen from 1 to 25 cases; in 9, from 25 to 100; in 2, this latter figure has been exceeded by Calvados, 112; Basses Pyrénées, 168. Of 6057 animals in the infected stables, 875 were affected (14.42 per cent.), 843 died. According to the reports of the sanitary veterinarians, 3219 animals of the bovine species were subjected to inoculation; of this number only 12 died (0.37 per cent.). (Tisserand: Rapport sur le service des épizooties en 1887.)—N. D. T.

inoculated 15,137 subjects in the canton of Berne alone. The results have been very satisfactory; the mortality from symptomatic anthrax has diminished considerably. In two infected communities the mortality was eight times less in inoculated animals than in others; in another it was five times less.

In Austria during the year 1885 Sperk inoculated 925 animals, which were afterward driven to the pastures in the Tyrolean Alps, which were notoriously infected. The results were excellent. None of the inoculated animals died of symptomatic anthrax (only 3 calves died after the first inoculation). Of 6387 non-inoculated animals anthrax claimed 107 victims. In 1886 they inoculated 2140 young oxen in the district of Salzburg and 3820 others in the Tyrol-Vorarlberg; the losses amounted to 4 and 16 animals respectively. Among the non-inoculated animals, 86 out of a total of 9160 died from anthrax in the first region and 330 out of 17,401 in the second. The mortality of the non-inoculated animals was from 10 to 20 per 1000; that of the inoculated, from 2 to 4 per 1000. The expenses of inoculation varied from 60 to 90 centimes (12 to 18 cents per head). In the Salzburg district in 1887, 2472 inoculated and 3571 non-inoculated animals were driven into infected Alpine pastures. The loss was 63 per cent. in the latter; in the others, 6.3 per cent., that is to say, 10 times less.

Of 963 animals inoculated in Baden during the year 1886, none died from anthrax.

In Prussia in 1886 they inoculated 64 oxen without any loss; in 1887 they inoculated 485, which were placed near 264 non-inoculated. Two inoculated subjects and 3 non-inoculated died from anthrax; the mortality has been 3 times greater in the latter. In 1888, 646 animals were inoculated and placed near 437 others. During the summer 4 of the non-inoculated animals died from symptomatic anthrax, while all the inoculated subjects resisted.

Kitt has observed that immunity may be conferred upon the ox and sheep by a simple injection of the virus exposed for six hours to a temperature of 85° to 90° C. The efficiency of this virus is almost equal to that of the second virus of the French experimenters. It is without danger for the ox. It may be injected into the subcutaneous connective tissue of the shoulder. The injection of a dose ten times greater than that which is required to give immunity is without danger. The local alterations produced by inoculation are insignificant.

INFLUENZA OF THE HORSE (TYPHOID FEVER).

Horse Disease: Red Epizooty: Intestinal Infection.

The term *influenza* was first used in human medicine to designate a general infectious disease accompanied by a catarrhal affection of the mucous membranes. From an etymological standpoint, this expression, which is of Italian origin, is synonymous with *epizootic strangles, distemper*. In equine medicine it served for a long time as a generic denomination which was applied to all infectious diseases the nature of which was unknown, but it was specially used to designate "a general pyretic disease with multiple visceral localizations which appear simultaneously in a certain number of horses" (extract of an opinion given by the consulting staff of the Berlin Veterinary School to the Royal Cabinet). Influenza has also been called: *fever of the horse, epizooty of the horse, nervous fever, putrid fever, typhus fever, typhus, catarrhal fever, infectious pneumonia (brustseuche, lungenseuche), red fever*. The diversity of symptoms that appear in diseases which are confounded with influenza has led to a recognition of different varieties of this disease; the following forms have been described: the *catarrhal, gastric, pectoral, red, typhus rheumatic, bilious*, etc.

Falke was the first who tried to clear up this confusion characterizing the affections which were designated collectively by the expression influenza. In his work, which was honored by the Academy of Medicine of Brussels, he recognizes two principal morbid states, of which we may always make a differential diagnosis. For the first, which is identical with the affection designated as the *disease of the horse*, by Dieckerhoff, and with Schütz's *red disease*, he has reserved the name *influenza*. The other, which corresponds with *contagious pneumonia* of Dieckerhoff, is described under the name *typhus*.

Dieckerhoff has shown recently that the division made by Falke is well founded, and that the diseases described by that author constitute two clearly characterized different morbid varieties. He has designated these under the names *horse disease (equine distemper) (pferdestaupe)* and *contagious pneumonia (brustseuche)*, and abandoned the name influenza. Friedberger has proposed to maintain the name *influenza* of Falke and to abandon *contagious pneumonia*.

We think it best to preserve this classification, for the following reasons :

1. Historically influenza is the older of the two diseases ; it represents the great majority of cases which were observed formerly.

2. From a practical standpoint we could not without inconvenience suppress the medical terminology of influenza, which deserves also to be preserved in remembrance of Falke's work.

3. The expression equine distemper (horse disease) does not give more information than that of influenza concerning the character of the disease, and there is an objection against it as a neologism.

4. It has been said that the expression influenza was still understood according to its ancient meaning by German veterinarians ; but the same criticism applies to the expression *brustseuche* (*contagious pneumonia*), which formerly was synonymous with influenza.

Influenza comprises :

1. *True influenza* [equine distemper (Dieckerhoff) ; *rothlaufseuche* (Schütz) ; *darmseuche* (Lustig)]. *Pferdestaupe* (Dieckerhoff).

2. *Contagious pleuro-pneumonia* (*brustseuche*). [We may distinguish contagious pneumonia of the horse or stable pneumonia, which constitutes an entire morbid entity, from the pulmonary localization of typhoid fever, which may exist in the same stable with other forms of this latter disease. The accidental introduction of a subject affected with typhoid pneumonia in the midst of healthy animals gives rise to cases of all forms of this disease, whereas that of contagious pneumonia is only propagated as pneumonia.—W. L. Z.]

HISTORY. Influenza is mentioned in several writings which are credited to the secretary of Charlemagne (Falke). According to Dieckerhoff, it was mentioned as early as the fourth century of our era. In the fourteenth century it was known in Italy ; in 1648, in eastern Germany ; in 1711, in the eastern provinces of Prussia. According to Heusinger, Löw¹ described it in 1729 as a disease of the horse which existed in an epizootic state in Italy, Austria, Poland, etc., and he has mentioned cases of transmission to man. It was seen in London in 1732 by Gibson. During the years 1760, 1776, and 1803, it existed in an epizootic state in dif-

¹ Löw : Febr. Katarrhal. a. 1729, historia.

ferent parts of England. Toward the end of the last century and at the beginning of the present, but especially in 1805, it spread extensively. At this period Havemann (Hanover), Naumann (Berlin), Wollstein, Pilger, and others published monographs of it which are quite complete; they considered mainly its symptoms and its course. Since then it has remained stationary in Europe, and particularly in Germany. It existed with special intensity during the years 1813-1815, 1825-1827, 1836, 1840, 1846, 1851, 1853, 1862, 1870-1873, 1881-1883. Among the ancient descriptions that of Auker, relative to the epizootic observed in Switzerland in 1826, is particularly remarkable. This author points out the contagiousness of the disease, and considers as the cause a volatile infectious agent. More recently the disease has been studied by Waldinger, Korber, Spinola, Hertwig, Hering, and Köhne; lastly, Falka gave a complete description of it in 1862.

In 1872-73 influenza appeared in an epizootic state in the greater part of the United States, where it was known as *pink-eye*. In France it is called *typhoid fever*.

The last European epizootic prevailed from 1881 to 1883. It was observed on nearly the whole Continent; it was seen especially in the large cities, and spread along the principal commercial highways. In Berlin it persisted for nearly two years; in Munich for eighteen months. From this period date the researches made by Dieckerhoff, Schütz, Friedberger, Siedamgrotzky, Lustig, and Vogel.

Etiology. Influenza is always developed after contamination. It has a more subtle contagiousness than any other infectious disease of the horse; it may affect a considerable number of them within a very short time.¹ In the stables propagation does not appear in spurts as in contagious pneumonia, it takes place regularly from one sick animal to its neighbors, and proceeds from one to the other. The infectious agent itself is unknown, but it must be extremely active. Without doubt it enters the system with the inspired air. It is contained in the exhaled air and in the excrements of the sick and convalescent (Friedberger). Outside of the organism it seems to lose its virulence rapidly. The receptivity of the horse for the germ of influenza is very great and is not influ-

¹ This is a statement quite absolute. On the other hand, there often develop two successive cases appearing in the two extremities of the stable. This fact is used as an argument by the partisans of the non-contagious theory.—L. T.

enced by age, sex, breed, hygienic care, or feeding. Dieckerhoff has contaminated healthy horses by injecting, under the skin and into their veins the blood of sick animals. Friedberger and Arloing, by proceeding in this way, have obtained only negative results. The transmission of the disease, as a rule, occurs by direct contagion (from horse to horse); it happens more rarely by certain intermediaries (grooms, litter, harness, thermometer, etc.). A first attack generally confers immunity for the whole duration of the epizootic (one to two years). Toward the end of the epizootic the cases become rarer and milder; this peculiarity is no doubt due to the lessened virulence of the germ.¹

Influenza is also observed in the donkey and the mule. Some cases of transmission to man and dog have also been related (Walther, Adam).²

Symptoms. After an average period of incubation of from four to seven days the disease appears. It is without prodromes and may reach its height within twenty-four hours.

It is generally limited to the circulatory apparatus, the nervous centres, the digestive and respiratory mucous membranes, the eyes, and the subcutaneous connective tissue.

1. Its first symptoms are loss of appetite, depression, and weakness. The temperature rises rapidly to 42° C.; this figure is sometimes exceeded; it remains stationary from three to six days, and is subject to only slight variations; then defervescence is produced as suddenly as elevation. Compared with the hyperthermia, the pulse is little accelerated; in the beginning we count from 40 to 50 pulsations per minute; later, from 60 to 70, and from 80 to 100 in cases where the disease is likely to terminate in death. On the other hand, when the temperature has returned to the normal point

¹ The duration of immunity is sometimes much longer. In nine horses of a stable which was infected by typhoid fever in 1889, four which had been affected in 1883 remained healthy (Champagne). In this particular case the immunity conferred by the first attack lasted six years. (See *Receuil Vét.*, 1890.)—N. D. T.

² Servoles and a few other veterinarians have affirmed the identity of typhoid fever of the horse with dothienenteritis or typhoid fever of man. This doctrine, which was combated by numerous authors, obtained but few supporters, and it has been definitely abandoned since Eberth's discovery. The pathogenic agent of typhoid fever of man has never been found in the lesions of typhoid disease of the horse (Nocard). These two diseases are only similar in name. "What has been called typhoid fever of horses does not at all resemble typhoid of man. The relationship which Servoles claims does not stand examination." (Chantemesse: *Traité de Médecine*, Paris, 1891.)

the pulse remains accelerated for some time. The febrile reaction is also remarkable by the irregular distribution of heat to the peripheric regions. Hyperthermia observed in subjects kept in infected stables, but which present the appearance of health, is a very important diagnostic symptom. It is an excellent measure to take the temperature daily of subjects which have been exposed to the contagion.

2. Fever is accompanied by considerable nervous depression and great muscular weakness. The animals hold their heads low and have the sleepy look of the immobile horse. They tremble while resting and stagger when walking; some are paralyzed in their hind quarters.

3. In a large number of cases the gastric symptoms predominate: the patients frequently gape; the buccal mucous membrane is red, dry, coated, and hot; when the buccal inflammation is propagated to the larynx we notice dysphagic phenomena. Colics are observed quite frequently; in the beginning constipation is the rule: the dung is hard and coated with a membranous or mucous layer (proctitis); later diarrhea occurs, which is usually accompanied by violent tenesmus; the feces are doughy, soft, or liquid; sometimes they have a fetid odor. The peristaltic movements are generally suppressed; exploration of the abdomen shows sensitiveness in some places. In the beginning, and notwithstanding the hyperemia, the urine has an alkaline reaction; it becomes acid as soon as the disease is localized in the intestine; it is rarely albuminous, but it contains, as a rule, a small quantity of desquamated vesical epithelium. In most cases micturition is very frequent (slight catarrhal cystitis).

4. The ocular disturbances, which are nearly always present, are characteristic. Ordinarily both eyes are affected. We sometimes observe a conjunctivitis, which is at first catarrhal; later, phlegmonous, with considerable tumefaction of the eyelids (chemosis), at other times a parenchymatous keratitis, and sometimes an exudative or hemorrhagic inflammation of the iris. The first symptoms are watering of the eyes, photophobia, a dark-red tint of the conjunctiva, and a contraction of the pupil. The eyelids, which are much tumefied, hot, and sensitive to the touch, remain closed. The conjunctiva is œdematous; it sometimes projects between the eyelids and forms a yellowish-red swelling. The sclerotic membrane often presents a yellowish tint; it also frequently forms a prominent ring around the cornea, which is of a grayish color. The lachrymal sacs

are filled with muco-purulent matter. The ocular globe is very sensitive to pressure; in the beginning the cornea is unctuous, iridescent, and sometimes seems dusted or smoked; later it becomes bluish or milky; its periphery is marked by a considerable vascular injection. The iris is hyperemic and tumefied; we may recognize hemorrhagic exudate in the anterior chamber of the eye. These inflammatory lesions ordinarily disappear with astonishing rapidity.

5. During the course of the disease we observe obstructions, which are due to cardiac weakness, and which become marked in the connective tissue of the extremities, the sheath, belly, and chest. They are cold, insensitive to pain, of doughy consistency, and present all the characters of passive œdema; it is only exceptionally that they become inflammatory or phlegmonous; when they exist in the legs the walk is stiff and restrained. In the great majority of cases resolution rapidly takes place and announces a cure. In some patients we observe an *urticiform* eruption.

6. The mucous membrane of the respiratory apparatus is the seat of a catarrhal phlegmasia, which is marked by a serous discharge in the beginning, and which later becomes muco-purulent by a slight tumefaction of the submaxillary lymphatic ganglions, a moderate acceleration of the respiration, and by a cough. We observe also a hyperemia of the vaginal mucous membrane. If the disease is somewhat prolonged, the animals become much emaciated.

Course and complications. The average duration of the disease is from six to ten days. In serious cases it is from two to three weeks; in benign cases, three to six days. At the stage of defervescence the appetite returns, the peristaltic movements are established and become more and more active, the general condition is ameliorated, the tumefactions disappear, and the cure is complete within eight to fourteen days.

In a small number of cases this regular course is interrupted by complications which are usually due to the hard work to which the patients are subjected at the initial period of the disease or during convalescence. These complications are:

1. *Pneumonia*. This may be developed when the respiratory mucous membrane is the seat of acute phlegmasia. Sometimes the pneumonia is catarrhal with a tendency to gangrene; at other times it assumes the croupous type, and then it generally becomes complicated with pleurisy. It is indicated by a great acceleration of

breathing and of the pulse by intense hyperthermia and by aggravation of the general condition. In some cases influenza is also accompanied by a phlegmonous tumefaction of the laryngeal mucous membrane, which is marked by serious dyspnœa and by contraction bruits.

2. *Cardiac asthenia*. The pulsation of the heart becomes more rapid, its action is palpitating; the pulse is accelerated, small, and then imperceptible. We also recognize dyspnœa and a passive hyperemia of the mucous membranes.

3. *Serious cerebral symptoms* and cerebral paralysis, or disturbances of medullary origin and spinal paralysis.

4. *Colliquative diarrhea* with fatal ending.

5. *Foundering*. This is due to the extension to the podophyllous tissue of the phlegmasia which is developed in the extremities.

Pathological anatomy. The principal alterations are found in the digestive apparatus. The stomachal and intestinal (especially that of the large intestine) mucous membranes (pyloric portion) are congested, tumefied, studded with hemorrhages, and deprived of their epithelium. They form thick swellings, which are translucent, of glassy aspect, and may attain the thickness of several centimetres; on incision a liquid escapes which forms small gelatinous masses. According to Schütz, Peyer's plaques are hypertrophied, inflamed, and softened, especially in the neighborhood of the ileocecal valve. In serious cases the submucous connective tissue is the seat of a yellowish infiltration. The buccal mucous and pharyngeal membranes sometimes show similar alterations.

The mucous membrane of the superior respiratory canals is marked by redness and catarrhal tumefaction; in some rare cases that of the larynx presents the lesions of phlegmonous inflammation (gelatiniform infiltration of the submucous connective tissue). This latter alteration is also found in the subcutaneous connective tissue on the surface of the regions where the skin is inflamed.

In the brain and spinal cord the subarachnoid spaces, especially the basilar surface, are filled with a liquid which is ordinarily clear, sometimes turbid, and rich in white corpuscles. The vascular plexus may also be tumefied and infiltrated; in one case the lateral ventricles contained quite a large quantity of liquid (20 cubic centimetres). As general alterations we may mention: the degenerative lesions of the principal organs (heart, kidneys, liver, spleen, and muscles), a slight hypertrophy of the spleen, hemorrhages of the

intestine, under the serous membranes, in the lungs, the eyes, and the brain; the inflammatory infiltration of the perirenal connective tissue and of the mesentery; tumefaction of the lymphatic ganglions; serous transudations in the large splanchnic cavities; imperfect coagulation of the blood and the slight consistency of the clots which it forms.

Prognosis. Influenza belongs to the group of benign epizootic diseases of the horse. The extent of damage varies with the season, period, and localities. Of 1700 horses affected by influenza, Dieckerhoff noted a mortality of 4 per cent. In 800 Aureggio found it 3 per cent. Friedberger has seen it reach 9 per cent., Siedamgrotzky 10 per cent. In the epizootic which existed in Philadelphia in 1872 30,000 horses were affected, and the mortality was 7 per cent. In the Prussian army it was reduced to 1.17 per cent. in 1886, and in 1887 (statistics referring to 1876 patients) to 0.85 per cent. In general, we see that the average mortality of influenza is 1 to 4 per cent. The greatest proportion of deaths is seen in the beginning of epizootics; the least, at their declining period.

Differential diagnosis. Influenza is distinctly different from all other epizootic infectious diseases in its rapid extension over large territories, its subtile contagiousness, its benign character, and by the abruptness of its appearance and the precipitous evolution of its symptoms. It is distinguished from contagious pneumonia by the predominance of its phenomena, which are due to the localizations of the process upon the digestive apparatus and the eyes; by serious neuropathic troubles; finally by the slightly affected condition of the respiratory apparatus. In contagious pneumonia symptoms of the pulmonary affection predominate, and attract attention from the beginning. However, at the period of invasion, when the influenza is not expressed by general symptoms (fever, inappetence, weakness, etc.), the differential diagnosis is sometimes difficult. Let us add that with these two diseases one does not by any means exclude the other; they may follow or accompany each other at the same time in the same animals.¹

¹ Under the denomination of alimentary infectious pneumo-enteritis Galtier and Violet have described a group of diseases which are confounded with typhoid fever and contagious pneumonia.

These diseases are indicated by a general chill and cooling of the skin. Their principal symptoms are: a lessening of appetite and general vigor; short-windedness and perspiration under the influence of slight exertions; a staggering, uncertain gait; an easy dropping of the hair of the mane and tail; a reddish-yellow, or yellowish-red,

Treatment. Influenza is but a slightly serious disease with typical course, which in the majority of cases does not require any therapeutic intervention. As in all other infectious diseases, the antipyretics are of little value; they may even exercise a harmful influence by disturbing digestion (salicylic acid, quinine, etc.). In cases where the process follows its normal course, diet, hygienic care, and a regulated ventilation of the stables are sufficient. When circumstances and the conditions of the atmosphere permit it, it is necessary to leave the animals at large, in the open air, in an isolated place, and where the healthy animals are not allowed to go. We may try to combat hyperthermia by means of cold rectal irri-

dark-red, or pale coloration of the conjunctiva; a more or less accelerated circulation (50 to 60 pulsations); a weak, irregular, or even intermittent pulse; strong contractions of the heart, which are also intermittent; lastly, an elevation of temperature, which oscillates between 39° and 40° C. In some cases the belly is in a painful state; the examination of the chest does not reveal any abnormal symptom. The duration of these morbid conditions varies from a few days to several months. Sometimes they end in a cure; at other times they become localized in one of the following organs: bronchi, lungs, pleura, heart (which is almost always affected), intestines, liver, spleen, kidneys, bladder, muscles, articulations, tendinous sheath.

The blood, urine, nasal discharge, intestinal matter, and all the tissues, having undergone alterations, are virulent. They owe their pathogenic properties to two micro-organisms at least—to streptococcus and diplococcus pneumo-enteritis equi. With cultures of these microbes we may reproduce pneumo-enteritis as surely as by making use of matter which has been taken from diseased animals.

Pneumo-enteritis is transmissible to the horse, sheep, rabbit, guinea-pig, and dog. Contamination, which is quite difficult through the digestive apparatus, occurs easily through the air-passages. In the goat, as well as in the horse, we may find cases of natural infection. Numerous influences predispose the horse to it: young age, weakness, transition from one season to another, colds, displacement and acclimation, insufficient or a poor quality of food, fatigue, excessive work, etc. The pathogenic agents come from the fodder, oats, or water. If they are ingested in a small quantity, they may not produce any disturbance; but in sufficient number, if the organism is debilitated, they determine pneumo-enteritis. They are especially dangerous when they penetrate into the air-passages. Immediate contagion is rare. A first affection does not confer immunity.

The prognosis is generally of slight gravity. However, in some cases, the mortality is quite serious; it has reached 25 and even 60 per cent. of the patients. A temperature of 41° C. and 80 pulsations are alarming prognostic symptoms.

We must see that a good quality of oats and other alimentary matter is given, the fodder must not be shaken in the stable, the oats should be dusted or shaken in the sieve, and the food be moistened with slightly-acidulated water (mineral acids, $\frac{1}{2}$ –1 per cent.); give as pure drinking-water as possible, or, if required, boil it; such are the principal indications of the treatment. While contagion from the sick is little to be feared, it is nevertheless prudent to isolate the patients and to disinfect the stable (phenicated water at 5 per cent.), to keep it well ventilated and perfectly clean. (See Galtier and Violet, *Journ. de Lyon*, 1889–90, and *Les pneumo-enterites infectieuses des fourrages ou variétés des affections typhoïdes des animaux solipèdes*. Paris, 1890.)—N. D. T.

gations, which besides have the advantage of re-establishing the peristaltic movements.

In serious cases and when complications occur active intervention is necessary. We have to combat cardiac asthenia with alcohol, camphor, digitalis, caffein, atropin, hyoseyamin, and veratrin. The gastric and intestinal localizations should be treated by alkalies (sulphate of soda, bicarbonate of soda, hyposulphite of soda); ophthalmia, by moist heat and atropin; the cutaneous tumefactions by frictions of camphorated alcohol. We should counteract intense hyperthermia with phenacetin, antipyrin, or antifebrin. The prophylaxis indicates the separation of the sick from the healthy animals, and the thorough disinfection of the stables.

Lustig, Spinola, and several other authors have advised measures of sanitary police. With Dicckerhoff, we think that it is not possible to resort to severe measures without serious inconvenience from an economical standpoint. It would, besides, be superfluous, considering the mildness of the disease. An obligatory declaration, which is recommended by Lustig, would however be a good measure, in the sense of attracting the attention of the public to the contagious and epizootic character of the disease.

CONTAGIOUS PLEURO-PNEUMONIA OF THE HORSE (BRUSTSEUCHE DES PFERDES).¹

NATURE. This pleuro-pneumonia of the horse is a contagious disease in which the pleura and the principal parenchyma (heart, kidneys) participate, as a rule, in the morbid process.

HISTORY. Contagious pleuro-pneumonia of the horse is the second of the diseases which formerly composed the nosological group designated under the name of "influenza." It has frequently been described and qualified as "*influenza pectoralis*," or as "inflammatory influenza form." It has also been given the names of "nervous pectoral epizooty" (*nervous Brustseuche*) and of "malignant pulmonary epizooty" (*bösartige Lungenseuche*).

Compared with the publications concerning influenza, those which we possess on contagious pleuro-pneumonia are of relatively recent origin. By examining the ancient clinical facts which were reported

¹ It is important to notice here that thus contagious pneumonia, which is also called pneumonia of the stable, is an entire morbid entity absolutely distinct from typhoid fever, with which it may be confounded.—L. T.

under the title of *influenza*, and laying aside those which refer to complications of this complex morbid state, we see that the first exact monographs of contagious pleuro-pneumonia date from the period when most of the veterinary journals were founded (1838). However, we do not find any relation of contagion of this affection as exists in influenza.¹

Etiology. The infectious agent of contagious pleuro-pneumonia is a small ovoid bacterium (Schütz), which appears mostly as "diplo-bacterium," a form due to the transverse segmentation of the microbe. It takes the methyl-blue well and all the original aniline colors; it becomes decolorized with Gram's method (coloration with aniline water and gentian-violet, and by the iodo-iodurated solution, decoloration by absolute alcohol); sometimes it presents an areola, which is colorable or not according to the case. The cultures, which are started by puncture in peptonized gelatin, appear as small, white, spheroidal islands, which do not liquefy the gelatin and do not become extended on its surface. In meat bouillon they constitute whitish flakes, which accumulate at the bottom of the bulb; on agar they form a turbid layer of a grayish color.

The inoculations made with these cultures give positive results in mice, rabbits, pigeons, and guinea-pigs; they have no effect on pigs and chickens. In the mouse they produce a septicemia of typhic course, which ends in death within twenty-four to forty-eight hours.

In the horse they determine very pronounced effects (Schütz). The injection, which is made directly into the lungs with a Pravaz syringe, produces all the symptoms of contagious pneumonia; this follows its usual course, and the alterations which are observed at the autopsy are those of gangrenous lobular pneumonia with degeneration of the principal parenchyma. In the organism of inoculated animals we find the characteristic microbes.

The bacteria of contagious pneumonia exist especially in large numbers in the lungs and the pleural exudate. They are also found in the nasal discharge and in the expired air (Rust). Their development is arrested at a temperature which is below $+10^{\circ}$ C.;

¹ Among the French works which were published on this disease we must particularly mention those of Cagnat: *Archives Vét.*, 1884; Delamotte: *Répertoire de police sanitaire*, 1886; Benjamin: *Receuil Vét.*, 1888; and Cadeac: *Journ. de Lyon*, 1889.

moist heat (manure) stimulates their pullulation. At the present time nothing precise is known as to the duration of their vitality outside of the organism. It has generally been admitted that in the invaded organs they are destroyed within six weeks. But in the encysted pulmonary centres they may preserve their activity for a very long time.

Klebs, Eberth, Koch, Friedländer, Fränkel, Zäselein, Salvioli, and others, have found in the lungs of man pathogenic micro-organisms; according to the case, they were streptococci, diplococci, or encysted micrococci.

Peterlein, Perroncito, Brazzola, and Mendelsohn have found in the pneumonic centres of the horse micrococci which were disposed in the form of small chains and separated from one another by narrow, clear zones (Peterlein). Under the name of *Bacterium pneumoniae croupose equi*, Perroncito has described voluminous spheroidal or ovoid micrococci, either isolated or united two by two (diplococci), or in larger number, and which are generally found surrounded by a clear gelatinous zone. They are especially distinguished from micrococci of the pneumonia of man because they are pathogenic for the guinea-pig and the rabbit; moreover, their capsule is not colored by the same agents.

In all these researches the pathogenic influence of the indicated microbes has not been established by culture and inoculation.

In 1885 Lustig cultivated a micro-organism different from that of Schütz, and has considered it as the agent of contagious pneumonia. Later he has made inoculations with the cultures. In his bacteriological studies he has obtained six different cultures; it is the last—a yellow culture—which he considers the true one, it is this which contains the infectious agent of pneumonia. If it is inoculated into gelatin by punctures, it grows upon its surface and along the track of the puncture, taking the aspect of a nail. The development is limited on the surface. The cultures are formed by very small ovoid bacilli, which take the Gram stain, but they are especially easily colored with a saturated solution of dahlia. The inoculations which were made by Lustig with these cultures have given the following results: nine horses which were inoculated in the pectoral cavity all contracted contagious pleuro-pneumonia; two died; the pleuritic exudate of one of these, which was obtained during life, gave the yellow culture (No. 6). Four horses which had been inoculated previously in the pectoral cavity and five other

non-inoculated subjects were exposed to natural contagion ; the first four remained unharmed, while the others contracted the disease.

Schütz, in his researches, has never been able to find the micro-organism observed by Lustig, while he has found in more than one hundred horses the microbe which was described by him ; fifty-eight times he has made pure cultures from it. Baumgarten, nevertheless, still has doubts about the pathogenic rôle of the bacillus of Schütz.

Affected animals and pathology. Contagious pneumonia is most commonly observed in crowded stables, in the army, in remounting stations, the stock farms, the dealers' stables ; it is especially in such places that it exists in an enzootic state. It is more frequent in the city than in the country ; it seems to have a predilection for young or adult horses (five to ten years) ; but it is possible that the smaller degree of predisposition of old horses comes from a previous attack. A first attack generally confers immunity for several years, sometimes for the whole duration of life.

The receptivity of the horse for the germ of this disease is not so marked as for that of influenza. In 700 horses of a regiment, all the stables of which were infected, the number of patients was 250 ; 400 resisted contagion ; 60 had had the disease previously (Rust). The horses which were in good condition resisted better than the subjects with a weak constitution.

The contagion is especially propagated by remounted horses or by such as have remained for some time in dealers' stables. The transmission sometimes takes place directly (from horse to horse) ; it usually happens through certain intermediaries (owners, veterinarians, stablemen, healthy horses, dogs, garments, fodder, manure, strange stables) ; the disease may be contracted by a short sojourn in an infected stable. The convalescent animals are particularly dangerous ; they may remain contagious for weeks (gangrenous pulmonary centres). The "miasmatic" origin of the disease, without direct or indirect contamination, has not been demonstrated. It has been admitted that the contaminating germ can long be preserved in the state of "stable miasma," no doubt because the contagion exists frequently in badly-ventilated stables, where the floor is continually soiled by fecal matters and lochia. But there, like anywhere else, it is the consequence of direct or indirect contamination by a convalescent animal or one which appears to be healthy. The irregular propagation of the disease in infected stables consti-

tutes a good sign by which to differentiate it from influenza, which is transmitted regularly from the sick to their immediate neighbors.

In army stables observation has taught that the horses which are placed in corners are much more exposed to contagion than the others, a peculiarity which is no doubt due to the insufficient ventilation of the stables. In the beginning the contagion often attacks a large number of animals. In the army it extends rapidly to the different stables of the same quarters. It is not rare to see it spread throughout a whole regiment in the space of a couple of weeks. Once, within two days, it affected forty-two horses in one squadron alone. The enzootic may be considered as over, five to six weeks on an average, after the last case has been observed. But frequently it persists for a long time; in certain collections of horses it is stationary. The permanence of the disease in these cases is due to the fact that subjects which are affected by chronic pneumonia (pulmonary cavities) remain with the others, or to successive infections which are occasioned by the importation of subjects healthy in appearance but which nevertheless carry the germs (remounting stations).

The principal means of entrance of the infectious agent is the respiratory apparatus, where it is introduced with the inspired air. It is also probable that the infection may take place by the digestive apparatus.

It is generally admitted that the duration of the incubation is about fifteen days; in some subjects it is shorter; in others a little longer. It is besides clinically impossible to determine it in a precise way, for we never know exactly at what time contamination has taken place; such are the contradictory indications which are given on this point. Perhaps the period of incubation is often much shorter. It may besides not be fixed by the lapse of time comprised between the period when the animals are placed in the infectious centre and when the first symptoms appear; it commences, in fact, only at the time of contamination.

Anatomical alterations. In the *lobular* form of contagious pleuro-pneumonia, which is by far the most frequent, we observe multiple centres of gangrenous pneumonia, with secondary pleurisy and parenchymatous degeneration of the principal organs.

1. In the lungs, particularly toward the base and the inferior regions, we find zones of hepatization; on section they show a dark red color, with a few clear grayish islands, which are dis-

tinctly circumscribed, and which vary in size from a pin's head to that of a child. When of recent origin they are very small, reddish-gray (hemorrhagic centres), and surrounded by a whitish zone formed by leucocytes, which have emigrated (reactive or limitative inflammation); when old they are dried-like and of spongy consistency (dry gangrene); on their surface are formed, later, cavities which vary in size from the diameter of a pea to that of a hen's egg, which contain fragments of necrosed pulmonary tissue, and the walls of which are constituted by a conjunctivo-fibrous capsule. In the regions where the air has access there exist ichorous centres filled with a fetid, putrid, viscous liquid (pulmonary gangrene). Finally, we find purulent centres of somewhat considerable dimensions containing a whitish pus mixed with mortified pulmonary tissue (disjoining furrow which is formed around the necrosed portions). In some cases the necrotic lesions are deficient, notwithstanding that during life the symptoms which betrayed them were observed; this peculiarity can only be explained by degeneration and absorption of the elements which are affected by death. Both pulmonary lobes are more or less hyperemic and œdematous.

2. The pleura shows lesions of diffuse exudative inflammation. This phlegmasia generally proceeds from the necrotic centres situated on the periphery of the lungs; but it may also become apparent by the absence of these latter. Both pleural folds are hyperemic and overrun with hemorrhages, rugous, or covered with soft red granulations. They are coated with a thick or membraniform, bluish or yellowish exudate, which is very easily detached. The pleural pouch contains a more or less abundant quantity (30 litres and more) of a turbid, reddish-yellow, grayish-red, reddish-brown, or dirty grayish-green liquid, which contains in suspension numerous yellow flakes, forming a deposit of variable thickness. In some cases the pleuritic exudate is plainly purulent (empyema); in others it is bloody (hemothorax); pneumothorax is rare. When the exudate is abundant it pushes back and compresses the pulmonary lobes in the vertebro-costal channels. It is possible that the disease follows a chronic course, and that the exudate becomes organized; then we see the appearance of fibrous, villous productions on the surface of the pleura, and the lungs contract adhesions with the pectoral wall and the diaphragm.

3. In the other organs we find the lesions of parenchymatous

inflammation and fatty degeneration. The myocardium, which is grayish-brown and friable, presents at some points a clay-like appearance and certain symptoms of fatty degeneration. The liver is enlarged; sometimes it has an icteric tint and has undergone the same degenerative lesions as the heart. The spleen is also increased in size, flabby, and studded with hemorrhages. The lymphatic ganglions, more especially those of the bronchi and mediastinum, are hypertrophied and softened, and of a reddish-gray color on section. The muscles are flabby and of a brownish-yellow shade. The serous membranes are little altered; we notice only small hemorrhages in the sub-serous connective tissue and a slight inflammation of the endocardium. The blood offers the least marked alterations. In the yellow pulmonary centres, in the pleuritic exudate, in the spleen, the liver, kidneys, and blood, we find the specific bacterium.

4. The gastro-intestinal mucous membrane is often hyperemic, tumefied, and overrun with ecchymoses; at times it presents superficial ulcerative lesions; the epithelium is desquamated. The lymphatic follicles are tumefied, sometimes gangrenous. The bronchial mucous membrane is inflamed and infiltrated.

According to Schütz, the pathology of these alterations is as follows: The bacteria penetrate into the lungs with the inspired air and determine a lobular gangrenous pneumonia, accompanied by simple necrosis of the pulmonary tissue; when the agents of putrefaction are associated with the specific bacteria the lesions again take on a putrid ichorous character, and if the pyogenic microbes intervene they are accompanied by suppuration. From the necrotic centres the inflammation may radiate to the neighborhood and produce a secondary fibrinous pneumonia or zones of hepatization. The destructive process of which some of these centres are the seat may also progress toward the pleura and determine inflammation, even perforation, of this membrane. Finally, it is possible that the necrosed portions may become absorbed or encysted by the formation of a peripheric connective tissue capsule. The general infection has its source in the gangrenous centres. The process when considered in its *ensemble* is similar to that of the *epizootic disease of the pig*.

The anatomical alterations of the lobar form, which is much more rare than the preceding, are those of croupous pneumonia.

Symptoms. The clinical picture of contagious pneumonia is

too complex to give a general description of it. We shall first consider the symptoms proper to the disease itself; later we shall study those produced by these various complications. The beginning is irregular; in some cases the trouble is apparent from the onset; in others it is announced by prodromic phenomena, weakness, loss of appetite, etc. Its first symptom is a rapidly increasing fever, the initial stage of which is frequently accompanied by chills. The temperature rapidly reaches 40° to 41° C.; it increases quite often by 3° in a few hours; we may count from 50 to 100 pulsations per minute, and this latter number is sometimes exceeded. The acceleration of the circulation is generally more marked than in influenza. The pulse becomes gradually weakened; soon it is very small and the precordial impulse palpitating. We observe besides acceleration of the respiration and general muscular weakness (but always less marked than in influenza); the conjunctiva and the other visible mucous membranes have a red or reddish-yellow coloration. The appetite has almost entirely disappeared.

According to the character assumed by the pulmonary affection, it is expressed by manifestations forming an *ensemble* with two somewhat different clinical tableaux. We distinguish:

1. A *lobular* pneumonia, with a tendency to necrosis and pleurisy.
2. A *lobar* pneumonia with benign course, without tendency to necrosis or pleurisy.

I. Lobular Form.

This form consists essentially of multiple circumscribed pulmonary lesions, and is marked by a high infectious fever, by serious general symptoms, by cough, and an acceleration of the respiration, which becomes more and more laborious. When the pneumonic centres are very limited percussion does not indicate any dulness on auscultation; we perceive normal or exaggerated vesicular murmur. In these cases the diagnosis is difficult; the disease may be confounded with influenza.¹ But usually several pneumonic centres become confluent; then we observe an irregular dulness on the surface of the lower anterior portions of the lungs, immediately back of the shoulders, and in the left precordial

¹ A very characteristic symptom of this disease is the disproportion between the very intense fever and the mildness of the local symptoms. The pale yellow color of the mucous membranes is different from that of typhoid fever.—L. T.

region; in this dull zone the respiratory murmur is weakened. We observe at the same time a rusty discharge, which dries up around the nostrils, forming brownish-yellow crusts. In the course of time the cough becomes weaker and dyspnoea increases. This lobular pneumonia may be recognized from the third day.

The extension to the pleura of the inflammatory process is marked at the onset by friction bruits and by extreme sensitiveness of the pectoral walls. When an abundant pleuritic exudate accumulates in the pectoral cavity the zone of dullness increases, and is always limited above by a horizontal line. It may occupy the inferior two-thirds of the thorax, and may even rise above this point. On auscultation we notice the absence of the respiratory murmur over this entire region, and find bronchial wheezing toward its upper part. The respiration assumes the abdominal type.

The transformation of lobular pneumonia centres into cavities is marked on percussion by a tympanitic resonance or by the cracked-pot bruit. The encystment of pleuritic exudates is marked by the same phenomena.

As the exudate increases the breathing becomes more laborious and dyspnoeic; in the act of inspiration the nostrils become extremely dilated. The febrile cycle is sometimes typic, at other times atypic.

When the disease has a favorable issue we see a critical polyuria, and a sudden lowering of the temperature occurs from the sixth to the eighth day. After a convalescence of from two to four weeks the cure is complete. But we may only expect such favorable ending if there is neither a complication of pleurisy nor abundant pulmonary exudation. In serious cases death may occur within a week. It is generally the result of one of the complications which will be examined further on.

II. Lobar Form.

This benign form is distinguished by a typic course, by the extent of the zones of dullness, and also in the great majority of cases by the absence of pleural phlegmasia.

Pneumonia ordinarily starts in the antero-inferior portions of the lungs, and then extends to a vast territory. It is mostly unilateral; in two-thirds of the cases it is localized in the left lung. Of seventy patients under Friedberger's observation only eight were affected by double pneumonia.

Percussion of the thorax elicits: first, in the beginning a tympanitic resonance or a slight dulness; second, at the stage of hepatization, a complete dulness, with a very marked sensation of resistance (see Percussion), and on the borders of the pulmonary lesions a tympanitic resonance; third, at the third period this resonance everywhere replaces the dulness. These typical characters are not observed in abortive cases; here the symptoms furnished by percussion are vague and their duration ephemeral.

Auscultation permits us to perceive: in the beginning, crepitant râles; in the second period, bronchial wheezing during expiration; in the third, moist râles. In the healthy regions we hear a coarse vesicular murmur.

The respiration, which is accelerated and difficult, assumes, as a rule, the costal type; the nostrils are greatly dilated; we may count from twenty to sixty respirations per minute. The expired air is hot. In the act of expiration we frequently hear a peculiar clapping bruit which is particularly designated by Liantard under the name of "trickling bruit," and by Dieckerhoff by that of "cracking nasal sound;" this latter author ascribes it to a transient adhesion of the mucous fold of the false nostril to the opposite membrane. The patients are generally found standing with their legs apart; they remain immobile, avoiding the slightest movements; they rarely lie down, but then always upon the diseased side. We may also hear a superficial, short, painful cough; but it is absent in a large number of cases. In nearly one-fourth of the patients, especially in those seriously affected, or when the pneumonia takes a hemorrhagic character, we observe in the beginning a saffron or rust-colored discharge, which dries up on the borders of the nostrils, forming yellow crusts.

This lobar form of contagious pneumonia has a typical evolution; the periods of obstruction, hepatization, and absorption follow one another at intervals which are nearly equal. The thermic cycle is also typical. The disease reaches its acme on an average of five or six days, and then enters upon the period of defervescence. The dulness often disappears in a very short time; the bronchial wheezing is replaced by râles; the respiration is slowed and the cough becomes more hoarse. In general, cure takes place within from eight to fourteen days (see Croupous Pneumonia).

Complications. Besides the symptoms produced by pneumonia and pleurisy we may observe, during the course of the disease, a

series of morbid phenomena which are associated with the preceding, but which sometimes exist alone and give to the clinical tableau quite a peculiar physiognomy. Ordinarily the most marked are produced by lesions of the heart, meninges, or kidneys. In 117 cases of this infection the lungs and pleura have been found unharmed twenty-seven times.

The principal complications are:

1. *Parenchymatous inflammation of the myocardium.* This is one of the most frequent and the most dangerous. It is marked by a small, accelerated pulse, imperceptible toward the end, by palpitations of the heart, by great weakness, and by general troubles which are due to obstruction to the returning circulation, and particularly accentuated in the respiratory organs (dyspnoea).

2. *Serious gastric symptoms:* violent colics, diarrhea, bloody excrements, etc.

3. *Hemorrhagic nephritis*, which is marked by hæmaturia, by the presence of *tube-casts* in the urine, by paresis of the hind quarters and a general progressive anemia. We have seen patients die from this complication in cases where pulmonary phlegmasia had entirely disappeared.¹

4. *Pyemia* and *septicemia* consecutive to suppuration or to putrefaction of pneumonic centres.

5. *Convulsions of cerebral origin* and symptoms of meningitis (leptomeningitis), which are the result of bacterian invasion of the meninges ("nervous form of contagious pneumonia"). Sometimes the infection is marked from the onset and exclusively by cerebral symptoms. Generally death occurs very rapidly by cerebral paralysis.

During the course of contagious pneumonia we may also observe abundant hemoptysis, renal hemorrhages, tendonitis, obstinate or repeated inflammations of the tendinous synovial sheaths, foundering, exudative and hemorrhagic iritis (internal ophthalmia), epileptiform attacks, convulsions in the facial and trigeminal region, paraplegia, paralysis of the great sciatic, of the penis (very rare), chronic endocarditis and valvular lesions, broken-wind, dropsy of the subcutaneous connective tissue, pharyngitis, pulmonary phthisis, disturbances of nutrition, falling out of the hair, wheezing, roaring

¹ We must add that this form of nephritis is sometimes accompanied by anasarca, which is first located in the legs and later in the trunk. I have gathered several observations of this kind.—L. T.

(observed in 10 per cent. of the affected subjects and due to neuritis of the recurrent laryngeal), and deafness. Finally, the disease may be complicated with influenza, strangles, petechial fever, etc. (mixed infections).

Course. It is extremely variable; it may be typic or atypic, acute, chronic, or abortive.

1. The typic course is observed in animals with a strong constitution, affected by the lobar form or by the benign lobular form. In these cases the fever, having reached its height, remains stationary for five to eight days (continued fever); later it becomes attenuated and disappears with the symptoms of pleuro-pulmonary inflammation. The termination is generally favorable, but convalescence is longer than in influenza (two to three weeks). Regulated ventilation of the premises, sojourn in the open air, and complete rest have a salutary influence on the course of the disease.

2. The abortive form is seen in old horses. Its duration varies from one to five days. The process is arrested at the stage of obstruction. In some cases we observe but prodromic manifestations; in others the only important symptom is hyperthermia.

3. The atypic course, with a tendency to complications, is principally seen in weak animals which are placed in unhealthy, badly-ventilated stables, or in subjects which are kept at work in the beginning of the disease. It is constant in cases of pulmonary necrosis.

4. The chronic form of contagious pneumonia deserves particular mention. It is produced by caseous pulmonary centres, which finally become encysted. In these lesions the infectious agent may multiply for months, preserving all its virulence. In general, the patients are able to perform but little work; they become rapidly tired, lose flesh, and show symptoms of asthma. Quite frequently, however, vague and insignificant symptoms only are observed. When these pulmonary alterations communicate with a bronchial tube they constitute a source of infection which is very dangerous; at a certain time they may also determine acute accidents or even fatal complications, as is proved by an observation related by Schwarznecker. In a troop of cavalry where contagious pneumonia had disappeared for three months, this author saw a thirteen-year-old horse become affected by fatal pleuro-pneumonia as a result of violent exercise. This horse had always appeared to

be in perfect health, and, notwithstanding that its temperature had been taken every day during the epizootic, the slightest hyperthermia had never been noticed(?). At the autopsy an old caseous centre was found which contained numerous specific micro-organisms. The violent exertions had caused a rupture of the capsule which had been formed around this old lesion; the bacilli had penetrated the pleural cavity and produced an acute sero-fibrinous inflammation.

Diagnosis. When contagious pneumonia assumes an abortive type it is often very difficult, in the absence of pulmonary symptoms, to make a diagnosis. Thus, when it occurs in crowded stables it is an excellent precaution every morning and evening to take the temperature of those animals as yet apparently healthy; this practice, which is followed in the army, enables us to recognize the disease at the onset.

In order to distinguish this disease from influenza or primary croupous pneumonia, we may resort to a bacteriological examination of the nasal discharge and proceed in various ways (Hell):

1. Staining preparations with fuchsin or gentian-violet. But the recognition of ovoid bacteria or diplococci does not suffice of itself to establish the diagnosis. The cultures and inoculation are alone decisive.

2. We make cultures by punctures in peptonized gelatin. In cases of acute pneumonia these cultures are characteristic: they are small, white, spheroidal, finely granulated, and disposed in separate colonies; they develop easily at the temperature of the room, do not liquefy the gelatin, and do not extend on its surface. The microscopical examination shows them to be composed of numerous ovoid bacteria.

3. Cultivation of bacteria in the oven and in bouillon at a temperature of 35° C. White flakes are deposited at the bottom of the bulb, while the bouillon itself remains limpid and transparent (when it becomes turbid the virus has not been obtained pure). The flakes are formed by long microscopic chains, which are packed irregularly.

4. Bacteriological examination by Gram's method. The preparation, which has been dried or passed through a flame, is placed for two to three minutes in gentian-violet with aniline water; then for two to three minutes in a solution of iodine and iodide of potassium (1:2:300 of water); afterward for ten to fifteen seconds in a bath

of acidulated alcohol (alcohol, 100 parts; hydrochloric acid, 3 parts); finally, it is washed in distilled water.

5. Inoculation of mice, which die in from twenty-four to forty-eight hours from typic septicemia.

A bacteriological examination of the condensed aqueous vapor contained in expired air and of the cultures of this liquid rarely give a positive result¹

Prognosis. The mortality of contagious pneumonia varies with its periods. It depends upon the course of the disease, the constitution of the patients, and the care which they receive; also upon the quantity of virus introduced into the organism, and the anatomical form of the process. In the beginning of epizootic invasions it is always higher than in the decline. Its extreme figures are from 0 to 20 per cent. In 1887, in the Prussian army, out of 2341 patients, 88 died. The average mortality was 3 to 4 per cent. In the various army corps it has varied between 2 and 8 per cent. The prognosis of contagious pneumonia, which is more severe than that of influenza, is also aggravated by the fact that a certain number of patients recover only imperfectly; they remain affected by permanent disturbances of respiration. The process, which is revealed in the lungs, may occasion therein, besides the encysted necrotic centres described above, a chronic interstitial inflammation which produces thickening, induration of the parenchyma of the organ, and a contraction of the field of the hematosis. In other cases a chronic inflammation of the bronchial mucous membrane remains, which is accompanied by bronchiectasis and peribronchial sclerotic centres. Moreover, pleural phlegmasia frequently results in adhesions of the lungs to the ribs and diaphragm, to hydrothorax, or to empyema. All these morbid conditions lead to permanent dyspnœa and gradual diminution of strength; they constitute, in a great many cases, the principal cause of asthma. We must also take into consideration the secondary affections generated by complications of contagious pneumonia (chronic roaring, tendinous synovitis, paralysis, etc.).

¹ We must observe that these excellent means, which may be practised in schools possessing well-appointed laboratories, are not within the reach of practitioners. Fortunately, they are not indispensable. The conditions under which the disease is developed, the intensity of the fever (which is disproportionate to the extent of the local symptoms), and the color of the mucous membranes characterize it well enough from a clinical standpoint to prevent confusion either with distinct pneumonia (croupous) or with typhoid fever.—L. T.

Treatment. The antipyretic treatment has not the value which for a long time has been attributed to it. In contagious pneumonia, as in influenza, the fever frequently resists antithermics. Generally we must be satisfied with wrapping the body in cold bandages and giving baths of cold water. When hyperthermia becomes intense and continues at its height we may use phenacetin, antipyrin, or antifebrin (in doses of 15 to 25 grammes), digitalis, alcohol, ether, and camphor. We have often obtained favorable results by using digitalis in large doses (10 to 12 grammes of digitalis leaves *per dose*). Of alcoholic liquids, we should use in preference wine (1 to 2 litres *per dose*); camphor should be used as camphorated alcohol or oil (10 to 25 grammes in subcutaneous injections). As heart stimulants, caffen (2 to 5 grammes in subcutaneous injection), hyoseyamin (0.01 gramme), and atropin (0.05 to 0.1 gramme) are also agents which may be recommended.¹

Inflammation of the pectoral organs is combated by Priessnitz's compresses or by other cutaneous derivatives; we should also try to stimulate the functions of the skin by frictions of essence of turpentine (1 part to 10 of camphorated alcohol). For rubefacient applications with alcohol (1 part to 12 to 20) the oil of mustard is of great advantage (Dieckerhoff); we may make several consecutive frictions with this preparation. Cantharides ointment, croton oil, etc., must not be used. In order to stimulate the elimination of inflammatory products of the lungs we administer expectorants (hydrochlorate of ammonia, alkalies, stibiates). Absorption of the pleuritic exudation may be induced by diuretics and hypersecreting agents (digitalis, alkalies, juniper berries, acetate of potash, pilocarpin in a dose of 0.1 gramme to 0.2 gramme in subcutaneous injections). If the pleuritic exudate rises to a great height, we may puncture the thorax; but the results of this operation are less favorable than in rheumatismal plenrisy; in contagious pneumonia agents which produce inflammation penetrate constantly into the pleura. Serious intestinal disturbances are treated with small doses of calomel (1 to 4 grammes) or by alkalies (Carlsbad salts). If cavities exist in the lungs, we must use inhalations of cresol vapor, phenic acid, or essence of turpentine; internally we should give tar or essence of turpentine. To combat laryngeal roaring of recent origin

¹ The combination of salicylate of soda, iodide of potassium, and digitalis has given me excellent results.—L. T.

we make use of small doses of strychnin (0.05 gramme) or arsenic (Fowler's solution, 10 to 20 grammes per day).

The prophylaxis is of the greatest importance. We should immediately isolate the sick horses, as well as suspected subjects, and disinfect the stable thoroughly with a solution of sublimate or cresol, or with chlorine vapors. The stable where the sick horses are placed must be well ventilated (freedom in the open air is specially favorable); the manure and damp litter must be removed without delay; the floor should be disinfected every day with a solution of sublimate at 1 per 1000 or of cresol 3 per cent. During the summer it is important to have a moderate temperature in the stable; for this purpose we may remove doors and windows. The patients should be kept absolutely quiet. The convalescent animals should be the object of particular watchfulness in order to avoid contamination of healthy subjects; it is proper to keep them isolated and under surveillance for at least six weeks, and to take their temperature in the mornings and evenings. It is also advisable to remove animals affected with catarrhal or any kind of internal disease. Persons having charge of the animals must submit to sanitary measures: the utensils which they use, the clothing and the shoes, the thermometers, etc., must be carefully disinfected. Finally, horses which have been recently bought must be subjected to a quarantine of six weeks.

When circumstances do not permit of adopting the measures (prophylaxis and disinfection) just enumerated, it has been advised to produce contamination as rapidly as possible of all the subjects. For that purpose the patients should be disseminated among the healthy animals; the nasal mucous membranes of the latter should be rubbed with the bloody discharge of the affected animals, and the mucus may also be mixed with their food. In 1885 this artificial contamination was carried out upon the horses of a Prussian regiment. Only young animals and those which did not show any symptom of a former affection were inoculated. Out of 220 horses contaminated in this way 81 became sick within two to three weeks (20 were affected by pneumonia, 10 by pneumo-pleurisy, 11 by myocarditis, and 40 showed more or less intense fever). All recovered. In most cases the disease appeared on the tenth day; its duration varied from one to eighteen days; in 32 horses it had a benign and abortive course and disappeared in one to five days. The practical usefulness of this inoculation cannot be appreciated

at the present time. No case of death resulted from the experiment. It is probable that this favorable result was obtained by means of the complete rest to which the animals were subjected from the start.

PREVENTIVE INOCULATION. Observation having taught that immunity is produced by a former infection, practitioners have tried to confer it by artificial means. At the military veterinary school at Berlin, in 1888, thirteen horses were experimented upon. The inoculations were made with the bacterium of contagious pneumonia which was cultivated in bouillon at oven temperature. The virus was inoculated under the skin, into the veins, the trachea, and the lungs. In one animal it was made to enter the digestive apparatus by mixing cultures with the food. The following were the results of these experiments:¹

1. The inoculation of Schütz's bacteria could be made in different ways without danger for the animals.

2. Inoculation into the subcutaneous tissue produced extensive painful phlegmonous tumefactions, which disappeared within five to ten days without having had any influence upon the general system. But this method is not advisable; one inoculation alone does not seem to lessen the receptivity for later infections.

3. A few hours (six to eight) after the injection of the virus into the trachea a typic disease was developed, which had an average duration of twenty-four hours; it was characterized by hyperthermia, loss of appetite, cough, a slight acceleration of respiration and circulation, more rarely by chills. The cough persisted, as a rule, a few days after the disappearance of the other symptoms; 20 to 60 grammes of liquid virus were sufficient to produce this trouble.

4. By direct injection of the cultures into the pulmonary tissue more or less serious phenomena have been produced, according to the quality of the virus used and the superficial or deep location of the lesions of inoculation. Small quantities of virus injected deeply into the lungs produced only transient symptoms. The injections made superficially in the neighborhood of the pleura, and the injections of considerable quantities of bacteria made deep into the pulmonary tissue, produced serious troubles clinically and anatomically similar to those of contagious pneumonia.

¹ Veter. Bericht f. die preuss. Armée, 1887.

5. The introduction of pathogenic agents in the circulatory and digestive apparatus was without effect.

6. Repeated tracheal and pulmonary injections (two to five) conferred immunity. When placed in an infectious centre for six to twelve days the inoculated animals remained unharmed.

TUBERCULOSIS.

A. Generalities.

KOCH'S BACILLUS. Tuberculosis is an infectious disease produced exclusively by the *Bacillus tuberculosis* discovered by Koch in 1882. This bacillus is a fine rod of an average length of $2-5\mu$ (nearly two-thirds of the diameter of a red blood-corpuscle), with rounded extremities. It is easily colored by aniline dyes, and may be cultivated, at the temperature of the organism, in gelatinized serum of the blood of oxen. Within ten days the cultures form small striations and whitish points which attain the size of a poppy seed within three or four weeks. The reproduction of the bacilli is performed by transversal segmentation; they generate ovoid spores, which are very resistant to destructive influences and which produce rods in their turn. The culture when inoculated in animals produces a typical tuberculosis. At the bacteriological examination of the lesions we find the bacilli between the cells of the tubercle and in their interior.¹

[The accompanying illustration show forms of tubercle bacilli observed in agar-agar cultures by Prof. Samuel G. Dixon, M.D. (*Medical News*, October 19, 1889), and in the liver of the green



jay of Mexico—*Xanthoura buxosa* (*Proceedings of the Academy of Natural Sciences of Philadelphia*, February 21, 1893). The same forms were found in human sputum by Prof. Allen J. Smith, M.D.,

¹ Nocard and Roux have recognized that bouillon and gelose to which $\frac{1}{2}$ per cent. of glycerin is added constitute an excellent culture medium for the tuberculous bacillus, especially for the aviary bacillus.—N. D. T.

in 1891, and have since been discovered by Klein, Henen, Fischel, Mafucci, and others.—W. L. Z.]

Anatomically, tuberculosis is characterized by the existence of cellular centres, which are round and without bloodvessels (*tubercles*). The histogenesis of the tubercle has been thoroughly studied by Baumgarten. When the bacilli penetrate into any kind of tissue they determine a fragmentation of cellular nuclei (karyokinetic segmentation) and a proliferation of the fixed elements of the tissue. The immediate consequence of this process is the formation of a large number of epithelioid cells with one or two nuclei, and which gather in more or less voluminous masses; these cells constitute the tubercles (tubercles with large cells).

These cellular elements are separated by a few rare connective-tissue fibres (reticulum of the tubercle). Sometimes the epithelioid cells acquire considerable dimensions and contain several nuclei (giant cells). The cellular proliferation is accompanied by an immigration of white blood-corpuscles; when the number of these latter exceeds that of the epithelioid elements we say that the tubercle is *lymphoid* or has *small cells*.

When it has reached the end of its development it has the aspect of a small translucent node of gray color and the size of a millet-seed (miliary tubercle). The tubercle being completely non-vascular, its cells undergo a series of retrogressive metamorphoses (caseous transformation or calcification).

The resistance of the virus of tuberculosis to the many destructive agents is due to the presence of spores. But the bacilli themselves are also endowed with great vitality; they preserve their virulence in common water for 120 days (Cadeac). In the water of the river Seine they may live for 50 days at a temperature of $+8-12^{\circ}\text{C.}$, and for 17 days at one of $+15-18^{\circ}\text{C.}$ (Chantemesse). Human tuberculous expectorations have remained virulent for 179 days in one case and 226 days in another. Dried expectorations subjected to the action of a spray of steam have lost their virulence in 15 minutes; dry heat has rendered them harmless in one hour (Schill and Fischer). In milk the bacilli are destroyed by a temperature of 85°C. (Bang). The virulence of dried human expectorations is destroyed within 20 to 24 hours when they are placed in contact with 8 to 12 times their weight of a solution of sublimate at 1:1000–5000. A solution of iodide of potassium at 5 per cent., iodoform in the state of steam and in powder, steam of iodine, lye

of soda at 1 per cent., phenicated water at 5 per cent., have the same action as sublimate. On the contrary, a concentrated solution of chloride of sodium, bromine water at 1 per cent., a watery solution of iodine (1 : 500), iodoform mixed with oil or essence of turpentine (Schill and Fischer), alcohol, putrefaction, disinfection, congelation, a temperature of 8° C. (Galtier), have no influence upon the tuberculous virus.¹

GENERALITIES UPON THE PATHOLOGY OF TUBERCULOSIS. The mode of infection of the organism and the evolution of the tuberculous process are not the same in all the animal species; they specially present great differences in cattle, pigs, and poultry. In this chapter we are compelled to restrict ourselves to general considerations.

It is principally through the lungs and intestines that the tuberculous bacillus reaches the organism. In the beginning tuberculosis is almost always a local affection developed in the organ through which the bacillus has invaded the system (lungs, intestines). However, it happens somewhat frequently that the organ which has given passage to the pathogenic agent remains unaltered, and that the morbid process starts in the neighboring ganglions (bronchial or mesenteric), and extends later to the pleura and peritoneum. Whatever may be the point where the bacilli are arrested, they determine first a formation of miliary tubercles which by gathering constitute pyramid-like masses more or less voluminous. Sometimes these infectious neoformations remain for a long time as localized inflammatory centres, or they become calcified; at other times they undergo a caseous degeneration or fibrous transformation; in some instances they suppurate, become ulcerous and produce cavities. When they become isolated by a connective-tissue proliferation from the surrounding parenchyma (lungs), or if they undergo complete calcification, the tubercles may heal. But in the majority of cases the process becomes general. The mechanism of this generalization is variable. It may take place:

¹ Dilution of tuberculous matters lessens their activity and may render them inert (Bollinger, Gebhardt); but the degree of activity of these matters varies considerably according to the mode of introduction. The animals generally resist the ingestion of contaminated milk when diluted in the proportion of 1 to 100, and even 1 to 50. The tuberculous expectorations diluted to 1 to 8 and ingested in a dose of 2 cubic centimetres often remain without effect; if they are inoculated into the subcutaneous connective tissue or the peritoneum, they must be diluted to 1 to 100,000 in order to render them inactive. By means of these latter modes of inoculation tuberculosis may be transmitted with diluted cultures at 1 to 400,000 (Roger, loc. cit.).

1. *By the way of lymphatics.* The ganglions which are next to the primary tubercles are first infected: in pulmonary tuberculosis the bronchial and mediastinal ganglions are involved; in intestinal tuberculosis the mesenteric ganglions, the spleen, and the liver; in tuberculosis of the upper respiratory passages, the subglossean ganglions, the upper and lower cervicals, etc. This ganglionic alteration is constant. Tuberculosis may be communicated to the serous membranes, especially to the pleura and peritoncum, by means of the lymphatics, starting from the neighboring ganglions which are the seat of an acute tuberculous process. It may pass from the abdominal cavity into the pectoral directly and without the intermediation of the bloodvessels: the bacilli cross the diaphragm by penetrating between the anatomical elements which constitute this wall.

2. *By continuity or contiguity of tissues.* This is a mode of propagation which is specially observed in the mucous membranes. A pulmonary tuberculous centre which opens into the bronchi may determine successively tuberculosis of the bronchial, tracheal, and laryngeal mucous membrane, etc., and deglutition of tuberculous secretions may in its turn generate intestinal tuberculosis. In tuberculosis of the kidneys or of the genital glands the process may extend to the genito-urinary mucous membrane.

3. *By means of the blood circulation.* The bacilli may be disseminated in the whole organism by the blood when the destruction of a tuberculous centre leads to perforation of a vein of certain calibre, or when the process directly invades the vascular walls; on the other hand, the lymph may carry it as far as the thoracic canal, and from there empty it into the anterior vena cava. The blood carries the bacilli into all the tissues, where they produce specific alterations. We distinguish two anatomical forms in the lesions which are thus produced: *a, chronic generalized tuberculosis*, which is characterized by isolated tuberculous centres and scattered in various organs; *b, acute miliary tuberculosis*, the development of which appears to be associated with the abundance of the bacilli in suspension in the blood, and which is characterized by the existence in all the viscera of a very large number of tuberculous granulations; the liver and spleen are always the first organs affected on account of their abundant blood supply. Besides, these two forms of tuberculosis often exist simultaneously.

The rare cases of *fœtal* or *germinal* tuberculosis are explained

either by direct infection which is of internal origin (the bacilli may traverse the foetal envelopes ; a demonstration of this fact has been given in experiments upon guinea-pigs and rabbits), or by the presence of bacilli in the sperm or in the ovule at the time of fecundation.

EXPERIMENTAL TUBERCULOSIS BY INJECTION OR INGESTION.

The first researches which were made on the infectious character of tuberculosis date back only twenty-five years. It was in 1865 that Villemin began the experiments which were to demonstrate the inoculability of the tubercle. By placing fragments of tuberculous matter of man under the skin and in the trachea of animals this experimenter has recognized that tuberculosis is an infectious specific disease, due to an agent transmissible by inoculation. Villemin also established the identity of tuberculosis of man and consumption in the horse. Klebs made inoculations into the serous cavities and has caused the ingestion of infectious matters ; he has affirmed the identity of bovine and human tuberculosis, and was the first to point out the dangers caused by the consumption of infected milk. Chauveau in 1868, by numerous experiments proved the transmissibility of tuberculosis by the digestive tract, and he has warned against the use of tuberculous meats. These observations have been confirmed by other French authors (Villemin, Saint-Cyr, etc.

In 1868, at the veterinary school of Hanover, Gerlach made experiments to demonstrate contamination by ingestion of tuberculous products. Out of 8 animals which had consumed these products and the milk of tuberculous cows 7 showed at the autopsy specific alterations of lymphatic ganglions, lungs, intestines, and liver. Gerlach has stated that milk and meat of tuberculous animals are infectious. Later, at the veterinary school of Berlin, he obtained positive results in 46 animals.

From 1870 to 1873 Günther and Harms made experiments on 94 subjects of various species with all kinds of tuberculous matter ; in 24 the results were positive ; in 70 they were negative or doubtful. The 14 experiments made at the veterinary school of Dresden in 1870 and 1871 have also given positive results for the greater number. The same has been the case in Zürn's, Bollinger's, and Roloff's researches.

The large number of negative facts which could be offered in opposition to these results caused us to reject for a long time the

possibility of the infection of man by tuberculous food. In 1876 the opinion of German veterinarians was expressed against that of Gerlach. But in the following years the positive results accumulated (Blumberg and Lange at Kasan; Orth, Toussaint, and Peuch; experiments which were made at the Berlin school from 1876 to 1880, at the Dresden school 1878-79). They related, besides, a great number of cases of transmission of tuberculosis to man and animals, by expectoration, milk, and infected meat. Finally, in 1882 R. Koch discovered, isolated, and cultivated the bacillus of tuberculosis. From that time the contagion of the disease was out of the question.¹

Baumgarten has always produced tuberculosis by feeding milk mixed with cultures of bacilli, and Bang has constantly obtained positive results by using milk coming from cows having their mammary glands overrun by tuberculous lesions.

In compiling the documents which were published from 1865 to 1884, Wesener gathered 369 experiments of infection through the digestive organs, with positive and negative results almost equal in number.

1. In 71 animals they tried to produce infection with human tuberculous productions; the guinea-pig and pig were the most sensitive animals to these matters.

[Dixon produced tuberculosis in the guinea-pig by injecting into the cellular tissues the juices of tuberculous matter taken from man, horse, cow, dog, cat, rabbit, opossum, hog, ostrich, chicken, and green jay.—W. L. Z.]

2. 180 experiments were made with tuberculous products coming from animals. In the calf, goat, and sheep the positive results have been the most numerous (in more than three-quarters of the cases); in the pig the proportion was from 3 to 4; in the rabbit, 1 to 2; in the cat it was greater than 1 to 3; and in the dog a little less.

3. The meat of tuberculous cattle has been fed in 32 experiments; of various susceptible animals, the pig has shown itself to be least

¹ From the first months of the year 1880 Toussaint had begun researches with the object of determining the pathogenic agent of tuberculosis. In making a culture of pulp of tuberculous ganglions he obtained cultures which were formed by small isolated and germinated granulations or gathered in irregular masses and constituted by a very refringent micro-organism measuring only 0.1μ to 0.2μ . He believed having at one time isolated the true microbe of tuberculosis (*Comptes rendus de l'Académie des Sciences*).—N. D. T.

sensitive to the infection. The dog is entirely refractory to it. Boiled meat has been found harmless.

4. The milk of tuberculous cows has been used in 86 experiments. Three-quarters of the pigs and one-half of the sheep and goats have become tuberculous. Boiling has lessened the virulence of the milk. Considered from the standpoint of their sensitiveness to tuberculous infection, the animal species should be classified in the following order: ox, sheep and goat, pig, rabbit, guinea-pig, cat, birds (with the exception of the chicken). The most active infectious products were tuberculous material from animals, expectoration of man, milk, and finally meat.

[Dixon reports a case of a dog which contracted tuberculosis after having been fed for six weeks continuously on infected bovine lungs.—W. L. Z.]

The numerous negative results which were observed in these experiments of infection by the digestive apparatus, according to Wesener, are due to various circumstances. Often the tuberculous products used were without bacilli or contained but a small number of them. The caseous or calcified tubercles contain scarcely any, and in those of the consumption of the horse very few are found. The meat is very rarely virulent. The milk is only infectious when there is mammary tuberculosis. The virulence of tuberculous matter is often considerably lessened by putrefaction, and the very short time during which the bacilli remain in the stomach and intestine is an unfavorable condition to the infection. In many cases also the animals were not well selected; they belonged to species which were hardly sensitive to the tuberculous virus. Finally the influence of the gastric juices seems to attenuate the activity of the bacilli. Wesener has made a series of researches upon this latter point in which fresh or dried tuberculous expectorations which were putrefied or previously subjected to the influence of the various digestive juices were brought directly into the stomach or intestines of rabbits. Those more recent from Straus and Würtz have shown that the gastric juice weakens the virulence of the bacilli and kills them in six hours. But it is without any influence upon the spores; these may reach the intestine and traverse its mucous membrane without affecting it, reach the lymphatics and produce tuberculosis of the mesenteric ganglions (*Tabes mesenterica*). The most dangerous food from that standpoint is undoubtedly milk, which contains bacilli and spores; the meat is much less so; it is very rarely viru-

lent and contains but few bacilli, and never any spores; moreover, it is usually eaten cooked. Several attempts at infection made by Bollinger with juice of meat from tuberculous bovines have given negative results (guinea-pig). The experiments of Bang, Zschokke, and Bollinger have established the fact that the milk of cows affected by tuberculosis may contain bacilli and possess infectious properties, even in cases where the mammæ are not the seat of any specific lesion.¹

¹ Of late years new researches have been made by Bang, Arloing, Nocard, Galtier, Gebhardt, Kastar, Dixon, and many others, with the object of determining the degree of virulence of the meat and milk and the dangers of infection by consumption of this food. From these researches we may draw the following conclusions: 1, tuberculous meat is rarely virulent; 2, the muscular juice very exceptionally contains bacilli; 3, the milk of tuberculous cows with healthy mammæ is sometimes infectious; 4, it is always infectious in cases of mammary tuberculosis; 5, the virulent milk generally becomes inert if it is diluted with 40 to 100 times its volume of non-infected milk; 6, boiling of milk and thorough cooking of meat destroy its virulence.

For several years numerous attempts have been made with the object of obtaining immunity against tuberculosis and to check the evolution of the tuberculous process. On August 4, 1890, at the International Congress of Medicine in Berlin, R. Koch announced that he had found substances which were capable of rendering guinea-pigs refractory to tuberculosis, and even of arresting the morbid process when they were injected in subjects having already reached an advanced stage of the affection.

In inoculating a second time guinea-pigs which were affected by tuberculosis and which showed cutaneous lesions, he recognized that the effects of the second inoculation were much less marked than those of the first; they were limited to a local necrosis, and the bacilli did not show any tendency to generalization. This result seems to establish that the organism may acquire a certain degree of immunity through the tuberculous products; the author attempted to find if the disease could not be combated by means of matters derived from the bacillus. The substance which he has used in his researches—*Koch's lymph* or *tuberculin*—is a glycerin extract of pure cultures of tuberculous bacillus. Small quantities of lymph are sufficient to kill guinea-pigs suffering from tuberculosis; with very small doses we may provoke more or less intense reactional symptoms, followed by marked improvement.

Koch's lymph is a limpid, brownish liquid, which is inactive when it is introduced into the digestive passages, but the effects of which are considerable when it is injected into the blood or the subcutaneous connective tissue. Its degree of activity varies according to the species and according to the healthy or diseased condition of the organism. It determines intense reaction in tubercular animals. In the healthy guinea-pig it may be injected without danger in a dose of 2 c.c.; in the healthy man 0.25 c.c. provokes serious trouble. In tubercular animals, a dose of 0.004 c.c., 0.003 c.c., or even that of 0.001 c.c., produces within four to five hours chills, and one, two, or three degrees of hyperthermia, fatigue, cough, sometimes nausea and vomiting. It possesses a very remarkable action upon tubercular alterations; under its influence the peritubercular tissues become inflamed and the seat of an exudation or abundant leucocytic infiltration; sometimes the neoplastic tissue resists, sometimes it becomes necrosed, but the bacilli which are contained within are not destroyed. In general, successive injections produce reactions which are more or less violent. If Koch has not as yet succeeded in isolating the active principle of tuberculin, he has obtained its purification by means of alcohol at 60 per cent. The action of pure tuber-

[In the *Medical News* of October 19, 1889, Prof. Samuel G. Dixon, M.D., announced the fact that he had produced immunity to tuberculosis in the lower animals by injections of devitalized tuberculous matter, and advanced the following hypothesis:

"1. It is possible that, by a thorough filtering out of bacilli from tubercular material, a filtrate might be obtained and attenuated so that by systematic inoculations a change might be produced in living animal tissues that would enable them to resist virulent tubercle bacilli.

"2. To bring about a chemical or physical change in living tissues that would resist tubercular phthisis, it is possible that inoculations with the bacillus would have to be made; yet before this could be done the power of the virulent bacilli would have to be diminished, otherwise the result would be most disastrous."

Dixon has also injected germicides directly into the spleen, and the constituents of the bile into the cellular tissues, the results of which are yet *sub judice*.—W. L. Z.]

culin upon the guinea-pig is about fifty times greater than that of the original product, and in man forty times. From a diagnostic and therapeutic standpoint the phenomena which are produced by these two substances are identical. Tuberculin has not given the therapeutic results which had been expected. It exposes the subjects to great danger; it frequently produces very intense and phlegmonous phenomena; it may also mobilize the bacilli and determine new tubercular growth. The facts which were revealed by Koch must nevertheless be considered as a very important experimental discovery.

Other researches have also been made with the object of eradicating tuberculosis or conferring immunity against this disease. Hericourt and Richet have studied the effects of injections of the blood and hepatic tissue of the dog; Bertin and Picq, also Bernheim, have used goat's blood; Baumgarten, Grancher, and Martin, attenuated cultures; Dairembert, Hericourt, and Richet, cultures of sterilized aviary tuberculosis; Courmont and Dor have employed a liquid which was obtained by filtration of cultures of aviary tuberculosis in glycerin water. Some of these processes have permitted us to slacken the course of the morbid process or to confer a certain degree of immunity to the animals.

Under the name of pseudo-tuberculosis we designate those affections anatomically characterized by neoproductions presenting the macroscopical characteristics of tubercles, but which are not produced by the bacilli of Koch. Roger has divided them into four groups: 1, *Pseudo-tuberculosis by foreign bodies*; 2, *Pseudo-tuberculosis by animal parasites* [in the cat by the *Ollulanus tricuspis* (Leuckart), in the sheep by the *Pseudalius ovis pulmonalis* (Koch); in the calf by the *Strongylus rufescens*; in the dog by the *Strongylus vasorum* (Laulanie), in man by the eggs of the distoma (Mima)]; 3, *Microbian pseudo-tuberculosis* produced by the various micro-organisms, especially by the zooglea—zoogloeoid tuberculosis; we may mention such examples in man, in animals, and in birds; 4, *Non-microbial mycotic pseudo-tuberculosis*; in this group we have farcy of the ox, caused by a parasite approaching the cladothrix (Nocard) and the pseudo-tuberculosis of pigeons produced by the *Aspergillus fumigatus* (Dieulafoy, Chantemesse, and Vidal). Roger: *Traité de Médecine*, Paris, 1891.—N. D. T.

[Addenda to Experimental Tuberculosis.]

REACTION OF THE AMIDE-GROUP UPON THE WASTING ANIMAL ECONOMY.¹

In 1889 I demonstrated that the tubercle bacillus and its nidus, when injected into the animal economy, produced an effect before unobserved.

In former communications I have been quite indefinite in my statements as to the real character of this toxic agent, having only suggested that it might be the residue of the pabulum remaining after the bacilli had selected that which was necessary for their existence, or a digestive secretion of the bacillus; or, again, that it might be an excretion of this living organism. However, in my endeavor to determine the true nature of the active principle of the indefinite mixture that others have entitled "tuberculin," I produced a crystalline substance that at once suggested the amide-group: allantoin, glycocin, tyrosin, kreatin and kreatinin, taurin and cystin, etc. With this fact directly before us that the wasting economy, accompanied by a defective liver and weak excretory organs, is often loaded up with waste products, it was believed worth while to institute a line of physio-pathological experiments by injecting the respective members of the amide-group into tuberculous animals. Kreatin being at hand, I at once injected a small quantity of its solution into tuberculous and healthy small animals with as satisfactory results as are usually obtained with guinea-pigs and rabbits. However, owing to the fact that these animals do not give entirely satisfactory reactions, W. L. Zuill, M.D., Professor of Veterinary Surgery in the University of Pennsylvania, kindly offered to assist in carrying out a line of physio-pathological experiments on the larger animals. His clinical experience, particularly with tuberculous cattle, made his services especially valuable in this work. A line of experiments was, therefore, immediately planned, and Prof. Zuill began the experiments to test the physio-pathological action of the respective members of the amide-group, when subcutaneously injected into the tuberculous animal economy as well as into those in health, for control experiments. The re-

¹ Report by Profs. Samuel G. Dixon, M.D., and W. L. Zuill, M.D., D.V.S., Academy of Natural Sciences of Philadelphia.

port of Prof. Zuill on his work up to the present time is as shown in the following communication :

TO PROF. SAMUEL G. DIXON, M.D.

DEAR DOCTOR: I hereby submit to you the clinical results obtained from the subcutaneous injection of kreatin in tuberculous cattle. The experiments were made in accordance with our prearranged plan, and have extended over the last two months. The results obtained in these experiments more than fulfil my utmost expectations, and are in every respect identical with those which I have obtained with tuberculin. The physiological action of kreatin in tuberculous cows is so exact and identical with tuberculin that it is impossible to recognize a clinical difference. Its influence upon circulation and respiration is well marked in animals suffering with miliary tuberculosis of the lungs; but large doses of the drug do not react upon these organs should the disease be confined to the other tissues of the body.

The action of kreatin upon tuberculous tissues is intensely energetic, causing rapid necrosis of this tissue, giving it the appearance of having undergone a cystic degeneration. The cheesy degeneration of tuberculous tissue seemingly disappears, and its place is taken by necrotic cavities filled with serum, in which float threads and masses of the tuberculous structure more or less large.

August 13th. Experiment No. I. was made with $\frac{1}{25}$ of a grain of kreatin in a tuberculous cow, with no well-marked reaction.

27th. Experiment No. II. was made with $\frac{1}{6}$ of a grain of kreatin in a tuberculous cow, which caused an elevation of temperature from 101° to $104\frac{1}{3}^{\circ}$ F.

September 9th. Experiment No. III. was made with $\frac{1}{2}$ of a grain of kreatin in a tuberculous cow, and caused an elevation of temperature from $101\frac{1}{3}^{\circ}$ to $103\frac{2}{3}^{\circ}$ F.

21st. Experiment No. IV. was made with 1 grain of kreatin in a tuberculous cow, and caused a reaction in temperature from $102\frac{1}{3}^{\circ}$ to $105\frac{3}{5}^{\circ}$ F.

4th. Check experiment No. II. was made with $\frac{1}{6}$ of a grain of kreatin in a healthy cow, and no reaction could be observed.

9th. Check experiment No. III. was made with $\frac{1}{2}$ of a grain of kreatin in a healthy cow, and no reaction could be observed.

24th. Check experiment No. IV. was made with 1 grain of kreatin in a healthy cow, and no reaction could be observed.

Respectfully,

W. L. ZUILL.

The report is so satisfactory in showing a marked action of one of the amide-group on wasting animals when subcutaneously injected, that not only will the chemical work to determine the exact character of the definite crystalline substance obtained from animal tissues and artificially prepared culture mediums be carried on, but also a full line of physio-pathological experiments by the subcu-

taneous injection of the respective members of the amide-group into the animal economy, in the Bacteriological Laboratory of the Academy of Natural Sciences of Philadelphia, such as its facilities will warrant.

ACTION OF THE AMIDE-GROUP UPON THE WASTING ANIMAL ECONOMY.

In carrying out the physio-pathological experiments by the subcutaneous injection of the respective members of the amide-group into the wasting animal economy, kreatin was followed up by taurin, with the results shown in the accompanying report made by Dr. Zuill, Professor of Veterinary Surgery in the University of Pennsylvania :

OCTOBER 9, 1891.

TO PROF. SAMUEL G. DIXON, M.D.

DEAR DOCTOR: I hereby submit to you the clinical results obtained from the subcutaneous injection of taurin into tuberculous cattle.

The experiments were made as nearly in accordance with your request as was possible; however, the stock under my control for the purpose was not so satisfactory as I would have liked.

Experiment No. 1 A was made upon a full-grown heifer that was previously used for the purpose of testing the action of both tuberculin and kreatin, therefore you will not look for as marked reaction as would likely take place under other conditions.

Treated with $\frac{1}{2}$ grain of taurin. Temperature reaction from 102° F. at 10 A.M., to 104° F. at 7 P.M.

Control Experiment No. 1 A, unfortunately, had to be made with a steer, supposed to be healthy, and only six months old, which fact, for obvious reasons rendered the result less satisfactory than it would have been with a full-grown animal.¹

Treated with $\frac{1}{2}$ grain of taurin. Temperature reaction from 101.6° F. at 10 A.M., to 103° F. at 9 P.M.

Experiment No. 2 A was made upon the same tuberculous heifer as was Experiment No. 1 A, therefore you would not look for as high a reaction as that shown by the first injections, particularly when in such close succession.

Treated with 1 grain of taurin. Temperature reaction from 102.2° F. at 10 A.M., to 103.8° F. at 7 P.M.

Control Experiment No. 2 A was made on the same healthy cow as Experiment No. 2 of September 4, 1891.

¹ February 10, 1895. Post-mortem report of this animal shows it to have been tubercular. It had received an intra-peritoneal injection of a pure culture of tubercle bacilli four months previous to the date of this experiment, and which was supposed to have failed to produce the disease. The mesenteric glands only showed signs of tubercular invasion.—W. L. Z.

Treated with 1 grain of taurin. Temperature reaction from 101.8° F. at 10 A.M., to 102° F. at 2 P.M.

Experiment No. 3 A was made with the same tuberculous heifer that I have been using to test the reaction of both tuberculin and kreatin, which renders the animal less susceptible to the action of taurin.

Treated with 1½ grains of taurin. Temperature reaction from 101.4° F. at 9.45 A.M., to 103.6° F. at 7 P.M.

Control Experiment No. 3 A was made on a healthy cow, by treating her with 1½ grains of taurin. Temperature reaction from 100.8° F. at 9.45 A.M., to 102.2° F. at 6 P.M.

Hoping to have some new cattle for the next of the amide-group,

I remain, very truly,

W. L. ZUILL.

The physio-pathological experiments with kreatin on the healthy and wasting animal economy were followed up by others upon taurin with the results heretofore reported :

JANUARY 22, 1892.

TO PROF. SAMUEL G. DIXON, M.D.

DEAR DOCTOR: At your request I have used kreatin, taurin, the toxic agent you extracted from tuberculous lungs, and tuberculin, as diagnostic agents, with the following results :

Cow condemned for tuberculosis treated with 1 grain of kreatin. Temperature reaction from 101.6° F. at 11 A.M., to 102.4° F. at 6 P.M.

Same animal treated with 1 grain of taurin. Temperature reaction from 102.2° F. at 9 A.M., to 102.6° F. at 7.30 P.M.

Same animal treated with tuberculous toxic agent, prepared according to Dixon, ½ grain. Temperature reaction from 102.4° F. at 8.55 A.M., to 102.6° F. at 7.30 P.M.

Same animal treated with 400 mgs. Temperature reaction from 101.8° F. at 12.50 P.M., to 102.4° F. at 8.15 P.M.

The results from the reagents not being consistent with our former experiments on tuberculous animals, the cow was killed and posted so as to determine the exact condition. No tubercular lesions could be found in any of the tissues.—W. L. Z.]

B. Tuberculosis in Various Domestic Species.

I. TUBERCULOSIS OF THE OX.

HISTORY. Tuberculosis of the ox is one of the oldest diseases which has been mentioned in domestic animals. Moses,¹ in his laws, forbade the consumption of the meat of animals which were affected by "phthisis." We find numerous measures relative to this disease in the Talmud, and particularly in the *Gemara* (fifth century after

¹ Moses: Third book, chap. xxii.

Christ), where frequent mention is made of *kandi*, *timari*, which expressions are considered as synonyms of *tubercle*. During the eleventh and twelfth centuries tuberculosis was distinctly recognized by the Arabian rabbi Alfasi and the Hebrew doctor Mainonides. There exists a collection of Jewish writings upon tuberculosis which is designated under the name of *Schulchan Aruch*, and which date from the sixteenth century.

In the ninth century, in Franconian portions of Germany, the church laws forbade the use of meat of oxen or pigs affected by consumption. In 1370, in Munich, the sale of tuberculous meat was forbidden. The same measures were taken in Würzburg in 1343, in Passau in 1394, at Landshut in 1401, in Würtemberg in 1558, in the Palatinat in 1582. In 1677 twelve scholars of a Leipzig school died of tuberculosis contracted by the consumption of infected meat. In 1702 Florinus gives a description of the symptoms of this disease, which was generally designated at that period under the name of "French disease," on account of the relations which were supposed to exist between tuberculosis and syphilis, the latter being considered as the cause of the other (Sodomy). The expression "French disease" seems to have been used for the first time by Van Helmont. All the tuberculous animals had to be killed and their carcasses destroyed. In the Prussian countries the annual loss from this cause amounted nearly to 4000 dollars (Graumann, 1784). A non-observance of the sanitary police regulations was followed by severe outbreaks.

In 1783 the superior sanitary college of Berlin published *instructions* on the inspection of meat, in which it indicated the characters of the "French disease" and criticised severely the admitted relation between this disease and syphilis; the consequence of which was that the meat of tuberculous animals was again allowed to be eaten. In the same year Kersting (Hanover) formulated a similar opinion and advised the same measure in recommendation to the government of Mecklenburg-Strelitz, Franck (Baden) also advised to authorize the consumption of meat when the lesions were not extensive. In 1784, in a report addressed to the government of Mecklenburg-Schwerin, Graumann declared that the meat of tuberculous animals had no noxious property. Thus, in 1785, all the edicts which forbade the sale of tuberculous meat were repealed. The same measures were taken in Austria in 1788.

In 1816 Tscheulin established three degrees of tuberculosis which

were taken into account for the inspection of meat, and he advised the following means : For cases of the first degree it is sufficient to remove the tubercles ; in the second degree the affected organs must be destroyed and consumption of the non-affected meat allowed, which is to be sold at low prices ; in the third degree the whole carcass must be destroyed. Until recently similar measures were enforced in Southern Germany, Switzerland, Austria, France, Belgium, Spain, and Italy.

Numerous expressions have been used in order to designate tuberculosis. The following have been specially used ; *pulmonary phthisis*, *pommelière*, *pearl disease*, *French disease*, *syphilis*, *veneria*, *lymphosarcomatosis*, *disease of the ganglions*. Tuberculous cows were also called *taurelières*, and tuberculous animals in general *consumptives*. *Pulmonary tuberculosis* and *pearl disease* have long been considered different diseases ; their identity has been established by experimentation (ingestion of virulent matter) and by bacteriological studies.

Etiology. In tuberculosis we recognize as the only cause the penetration into the organism of Koch's bacillus. The mode of infection is variable. It is mostly produced by cohabitation. Cases have been recorded in which the introduction of a tuberculous subject into a stable until then not infected resulted in a contamination of all the neighboring animals. Contagion may take place by the intermediation of expired air or by tuberculous expectorations. In the calf it is frequently produced by milk, especially when the mammary organs are the seat of specific lesions. Quite a considerable number of observations demonstrate the existence of congenital tuberculosis which is produced by a direct passage of the bacilli from the mother to the foetus (foetal tuberculosis, or "germinal"). Cases of infection in the first months of intra-uterine life are not at all rare ; but generally the females abort early. The infection produced at the time of copulating may be exceptional, but does not appear to be established. Nothing is known of contamination of cattle by man ; some observations only have been mentioned where swill and kitchen refuse appear to have been the agents of infection. The possibility of this fact is not to be doubted of having conveyed the contagion of tuberculosis of man to other animal species, to the chicken for instance. Lydtin has mentioned a case of transmission of tuberculosis by inoculation of contagious peripneumonia ; the lymph which had been used for the inoculation came from a tuberculous animal.

The penetration of the bacillus into the organism may take place in healthy mucous membranes. Certain predisposing influences, which were formerly considered as essential causes of tuberculosis, favor infection; such are, mainly, insufficient, watery food, which is poor in protein (malts); also narrow, poorly-ventilated stables. The catarrhal affections of the respiratory mucous membrane favor the entrance of the bacillus, especially when the morbid secretions remain in the bronchi. In the SMOKE DISEASE OF THE ESTABLISHMENTS USING FURNACES, and which represents, according to Johne, true chronic tuberculosis by inhalation, the inspiration of smoke produces at first an inflammatory affection of the respiratory mucous membrane, which prepares the ground for the bacillus. The lacteal secretion and numerous gestations predispose also to tuberculosis by weakening the organism, rendering it less resistant to the attack of virulent elements. It is, therefore, not rare to see an aggravation of the disease after parturition and a great progress of the affection at the period of puberty. Consanguinity, selection, improvement of the breed, are also influences which favor the invasion of the system by the tuberculous agent. Finally, there seems to exist a native predisposition created by transmission of the weak constitution of the mother to the young.

[Dixon calls attention to the possible relationship between the tubercular diathesis and nitrogenous metabolism (*Therapeutic Gazette*, December 15, 1894).—W. L. Z.]

AFFECTED ANIMALS. Tuberculosis is the most common disease of the bovine species. It is found in nearly all countries, especially in the neighborhood of cities. In the polar regions, in Northern Sweden and Norway, in the steppes (Spinola), in certain islands which are far from the Continent, as Iceland (Krabbe), in Sicily (Chicoli), it is extremely rare, even unknown. It is no longer observed in wild herds (Veith). The proportion of tuberculous bovines varies according to the countries. According to Göring, in Bavaria, during 1877–1878 it was 1 to 2 per 1000. Lydtin found it 2 per 1000 in the Duchy of Baden (1881), but he thinks that this figure is below the reality. There are districts where tuberculosis is much more frequent. In the Hollandish breed the proportion of tuberculous animals is said to reach 50 per cent. (Nathusius). In Holland it is 20 per cent. (Schmidt); in the district of Grumberg, 15 to 20 per cent. (Wolf); in some countries of Hohenzollern, Pomerania, Bromberg (district of Uetze), 50 to 60

per cent. (Schantz, Allerechh); at Hildesheim, 60 to 70 per cent. (Haarstick).

The statistics obtained from slaughter-houses vary also according to the countries. In München and Augsburg the number of cases of tuberculosis varies from 2 to 11 per cent.; in Berlin it is 4 to 5 per cent.; in Chemnitz, 3 to 5 per cent.; in Strassburg, 2 per cent.; in Baden, 1 to 2 per cent.; in Bremen, 2 per cent.; in Paris, 6 per cent.; in Belgium, 4 per cent. The animals belonging to the so-called prairie or valley breeds (Hollandish) furnish a larger number of cases of tuberculosis than those of the mountain races, which have a stronger constitution. Animals kept permanently stabled are affected in greater numbers than those at pastures. Tuberculosis is particularly common in dairies and in those attached to distilleries, breweries, refineries, etc. It is more frequent in the cow than in the bull or ox, and in old than in young animals. The number of cases which have been recognized in the calf is very small because intra-uterine infection ordinarily leads to abortion. In one million calves which were killed in München from 1878 to 1882 only five cases of congenital tuberculosis were recorded. In Berlin the proportion was 7 per 80,000 during 1885–1886, and 6 per 90,000 in 1886–1887. In Augsburg, in 230,000 calves killed from 1873 to 1886 only nine were found to be tuberculous.

Pathological anatomy. The anatomical alterations of tuberculosis are generally found in the lungs and serous membranes (consumption, pearl disease). The statistics of Lydtin, Göring, and Adam have shown that the serous membranes and lungs are affected simultaneously in half of the cases, the lungs in only one-third, and the serous membranes alone in about one-fifth. The lymphatic ganglions which supply the diseased organs are always affected. In generalized tuberculosis all the viscera sometimes present specific lesions, at other times the morbid process invades but a few or even only one of them.

1. In the lungs we find as essential and primary alterations milary tubercles; these are small nodes of the size of a millet-seed or a pin's-head; they are quite consistent, of a yellowish-white color, and seem transparent in the beginning; they frequently exist in considerable numbers; sometimes they are disseminated in the perfectly healthy pulmonary parenchyma. The old tubercles contain a caseous or calcareous point in their centre; they are frequently gathered in more or less voluminous masses which undergo calcifi-

cation and caseous transformation throughout.¹ We find, besides, caseous pneumonic centres of variable dimensions, which are developed consecutively to catarrhal pneumonia and accompanied by ectasia of the alveoli and bronchioli, also by abundant leucocytic diapedesis and accumulation of large epithelioid cells inside of the alveoli (desquamative pneumonia). Later, the affected pulmonary areas undergo caseous or purulent destruction. It is in this manner that these are formed; cavities and centres are filled with yellowish, cheesy, viscous, or purulent contents. Besides these lesions we find scleral alterations of the interstitial tissue, which are specially localized in the neighborhood of caseous centres and miliary tubercles; they are marked by a connective-tissue neoformation and by hardening and atrophy of the pulmonary tissue; this tissue, which is very resistant to the incision, sometimes presents a cartilaginous consistency; it may even become calcified. Frequently the lungs also show accessory lesions (echinococci, contagious peripneumonia, etc.).

The mucous membrane of the bronchi is generally the seat of a chronic catarrh, and bronchiectasis is not rare. This membrane and that of the larynx often present miliary tubercles disposed in regular lines and ulcerations with thickened and indurated borders. The tubercles developed in the bronchi ordinarily overrun the peribronchial connective tissue (bronchitis and knotty peribronchitis) as well as the pulmonary parenchyma.

The bronchial ganglions are regularly tumefied and hypertrophied; in some cases they are found filled with miliary tubercles; in others, they have undergone caseous degeneration or calcification.

In the pulmonary pleura we sometimes recognize a chronic phlegmasia with adhesion of the lungs to the pectoral walls.

2. Tuberculosis of the pleura and peritoneum (pearl disease) begins by the formation of very small nodes of a transparent, light-gray color (the "pearls"), which are smaller than millet seeds, giving to these membranes a granular aspect; we observe in their periphery a congestion of the serous membrane and a connective-tissue neoformation; the "pearls" are then found encased in a fibrous framework. By the confluence of tubercles and connective

¹ Caseous softening is sometimes produced in the centre of extensive tuberculous masses and forms vast cavities, which may open into the bronchi and be partially emptied. They are distinguished from bronchial dilatations by the absence of epithelium in their wall.—L. T.

hyperplasia there are formed, upon the parietal fold as well as upon the visceral fold, irregular masses, which become larger and larger, and may acquire the size of a fist. In the beginning they are of soft consistency and of reddish-yellow or dark-red color on section; later, they become resistant, hard, and rugous; sometimes they acquire a fibrous texture and creak under the knife; sometimes they undergo caseous transformation or calcification; in their centre exists a friable island, which is yellowish-gray, grumous. At this advanced stage the tubercles have a yellowish-gray color, which is ashy or bluish-white. Their disposition is quite peculiar; they generally constitute polypoid masses, which are disposed like cauliflower or grapes, with a more or less wide basis; in some cases, they cover the serous membrane entirely and attain considerable dimensions (30, 40 kilogrs. and more). These tubercles are sometimes closely joined; at other times they form agglomerations which have a variable volume, and are united by connective-tissue bands.

The ganglions of the mediastinum, like those of the bronchi, are hypertrophied and studded with miliary tubercles or affected by caseous degeneration. They form large tumors, measuring as much as 50 centimetres in length; they often surround the œsophagus and compress it.

3. We find also tuberculous alterations in a large number of other lymphatic ganglions: the sub-glossean and the parotidi ganglions, the upper and lower cervical, the ganglions of the shoulder, of the axilla, elbow, groin, and stifte-joint; the sub-mammary ganglions, the external ischiatic, intercostal and sternal, the mesenteric and also the lumbar; the ganglions of the liver, spleen, kidneys, etc. All may show considerable enlargement.

4. In the abdominal cavity, the organs most frequently affected are the peritoneum (the parietal fold, epiploon, and mesentery), the lymphatic ganglions, the liver, and spleen. Tuberculosis of the spleen begins as a rule with a uniform tumefaction of the Malpighian corpuscles (follicular hyperplasia). The tuberculous granulations are developed in the lymphatic follicles and the perivascular connective tissue. Tuberculosis of the kidneys is quite common. These organs are hypertrophied, and contain numerous miliary tubercles which have become degenerated in the centre and are surrounded by a connective-tissue capsule. According to Schütz, these tubercles develop in the interstitial connective tissue and crowd the renal parenchyma back, which becomes atrophied. Sometimes we

find simultaneously a parenchymatous or hemorrhagic nephritis. The tuberculous ovaries have often the size of a man's head. The uterus may acquire enormous dimensions; its mucous membrane is generally strewn with ulcerations. The submucous subserous connective tissue and the muscular layer contain tubercles; when these are much developed they find an exit in the uterine cavity; sometimes we notice tuberculous degeneration of the cotyledons. Frequently the Fallopian tubes are obstructed by tubercular proliferation. In the vagina we may find alterations similar to those in the uterus. In incising the testicles we observe more or less voluminous tubercles and caseous centres. Tuberculosis of the vaginal sheath and hydrocele are not rare. The testicular cord, the different canals, and the prostate may also be affected.

The intestinal mucous membrane, more particularly that of the large intestine, sometimes shows tuberculous ulcerations; these are less frequent in the abomasum and the third stomach (Johne). In the stomach and intestine the submucous and subserous connective-tissue layers contain a large number of tubercles.

5. Chronic mammary tuberculosis is marked by a diffuse and uniformly hard tumefaction; the glandular *acini*, which are hypertrophied, have a grayish shade (Bang); on the sections made into the altered portions we observe small yellowish, linear or punctiform spots and ecchymoses; the large galactophorous canals contain yellowish caseous masses rich in bacilli. Later we find an abundant connective-tissue neoformation (chronic interstitial lobular mammitis) and nodes of various size and hard, which frequently undergo caseous transformation and calcification. The mammaræ, which are knotty and sometimes of stony hardness, may reach enormous dimensions (20 kilogrammes and more). In the walls of the galactophorous sinus and in the principal canals there exist numerous miliary tubercles; all the altered parts are infiltrated with bacillary accumulations. The lymphatic ganglions, which are located above the posterior quarters of the udder (supermammary), are hypertrophied, hard, and caseous.

6. The tuberculous lesions of the brain and of the spinal cord are more frequent than is generally admitted. In forty tuberculous cows Semmer has four times observed specific lesions of the brain. In the pia mater, the arachnoid, and cerebral substance there exist tuberculous masses which are more or less old, and from the size of a millet-seed to that of a hen's egg, with a coloration varying from

yellow to gray. They are mostly located in the pia mater and in the base of the brain, where they may exist for a very long time without producing any inflammatory alterations; frequently, however, they are accompanied by meningitis (*tuberculo-basilar leptomeningitis*). In some cases the meninges become joined; in others the process leads to internal hydrocephalus. Sometimes the tubercles become gathered in large masses. Similar lesions are found in the spinal meninges and in the spinal cord; in these may be observed atrophies produced by compression (Johne).

7. We also find tuberculous lesions in the eye (tubercles of the iris and choroid, transformation of the bulb into a caseous or granular mass), in the muscles (miliary or larger tubercles in the muscles of the hind quarters, abdomen, chest), in the myocardium and pericardium (cardiac symphysis), in the bones (temporal, occipital, cervical vertebra, spinous processes of the dorsal vertebra, ribs, sternum, or long bones). Bony tuberculosis begins, as a rule, in the medullary tissue; reddish-gray granular centres are formed in it; the bony substance undergoes here and there lacunary destruction (rarefying osteitis, destruction of the lamella), then the marrow undergoes caseous transformation and cavities are developed in it. The tubercular process sometimes invades the cartilages (nasal wall, conchinean cartilages of the ear, etc.), the articulations (*synovitis pannosa tuberculosa*), especially those of the hip, stifle, knee, and the periarticular tissues (*periarthritides tuberculosa*), the large bloodvessels (walls of the aorta), and the skin (tumors, ulcerations).

[Dixon planted bovine tubercle bacilli in the choroid of a chicken's eye and produced a localized tuberculosis of the eye. The bacilli were found alive at the end of the third year after inoculation.—W. L. Z.]

The general condition may be excellent in animals affected by very extended tuberculous lesions; this is specially common in "pearl disease." But advanced pulmonary tuberculosis is always accompanied by emaciation and anemia. In the last stage of the disease we also observe the symptoms of hydræmia.

In the Berlin slaughter-house R. Ostertag has made researches upon the pathology of tubercular lesions in the various organs, and has tried to define the meaning of the expressions: *local tuberculosis* and *generalized tuberculosis*. In the ox the various forms of primary tuberculosis result as a rule from an infection of the respiratory or uterine mucous membrane (pulmonary tuberculosis and

nterine tuberculosis). Quite frequently also tuberculosis of the serous membranes (pleura, peritoneum) may appear as a primary disease when the bacilli traverse the lungs or intestines directly without leaving any trace behind.

Primary tuberculosis of the uterus may extend to the peritoneum through the oviducts by continuity of the tissues, and afterward to the plenra through the diaphragm. Primary peritoneal tuberculosis leaves the parenchyma of the abdominal organs unharmed, with the exception of the Fallopian tubes and the liver (region of the portal system); it ordinarily becomes localized to the anterior parts of the abdominal cavity and extends in the direction of the lymphatic canals which emerge from the abdominal cavity—that is to say, from back to front. These lymphatic vessels start from the lower abdominal and lateral walls, cross the diaphragm, and penetrate into the pectoral cavity, where they reach at first the posterior mediastinal ganglions, then the anterior and bronchial ganglions. From these latter the infection may be communicated to the pleura, but the process does not pass from the plenra to the lungs (with the exception of the small portion of this organ situated on the surface of the hilum).

Primary tuberculosis of the pleura cannot be communicated to the peritoneum on account of the opposite direction of the lymphatic current; it also leaves the pulmonary parenchyma intact. Sometimes primary tuberculosis of the lungs produces infection of the pleura directly, at other times we observe from the very beginning tuberculosis of the bronchial ganglions. The same is the case in primary tuberculosis of the intestines, which may determine either tuberculosis of the peritoneum or infection of the mesenteric ganglions, which leads in its turn to infection of the peritoneum. Primary intestinal tuberculosis may also determine tuberculosis of the liver without the intermediation of the general circulation, and consequently without generalization when the bacilli are brought into the hepatic tissue by the portal system.

In the ox, no matter whatever may be the primary location of tuberculosis, when the disease becomes generalized the order in which the various organs are invaded is usually the following: lungs altered (changed in most cases equal to 100 per cent.), plenra and peritoneum (90 per cent.), liver (85 per cent.), buccal cavities, pharyngeal and intestinal cavities (60 per cent.), spleen (50 per cent.), kidneys (30 per cent.), os (5 per cent.).

In females the proportion of cases of tuberculosis of the uterus reaches 65 per cent.; for the mammæ it is 5 to 10 per cent.; for the ovaries, 5 per cent. Tuberculosis of the genital organs of the male is much more rare.

From the diagnostic standpoint of the *local* and *general* forms of tuberculosis, Ostertag gives the following indications which serve actually to regulate the Berlin slaughter-house.¹

I. Tuberculosis is *local* in a broad sense of the word.

1. When the peritoneum or the pleura and the corresponding ganglions only are affected.

2. When one or several organs of the same splanchnic cavity and the serous membrane which covers it are altered, lungs and pleura, intestines or uterus and peritoneum, intestines and liver only; finally, the intestines, liver, uterus, and peritoneum, without the other organs; the lungs seem to be the first affected.

3. When the disease of one only or of several organs of the abdominal cavity is accompanied by tuberculosis of the pleura.

II. Tuberculosis is *generalized*:

1. When there exist specific lesions of the pleura or of the peritoneum, or of these two serous membranes and *miliary tubercles* in *one organ* (lungs, for instance).

2. When these two serous membranes are affected and there exist embolic centres defined in different organs (lungs and liver, lungs and uterus, etc.).

Symptoms. Tuberculosis of the ox is slow in its development and chronic in its course. Its beginning is insidious, and escapes observation; in only a few cases is it betrayed by pyretic phenomena. The manifestations vary with the localization of the disease.

1. *Pulmonary tuberculosis* is marked by a slight, short, deep, spell-like cough, which becomes extremely painful later and is

¹ *Tuberculosis of the spleen is always a symptom of generalization of the infection; this organ only becomes tuberculous when the bacilli are brought into it by the general circulation.* Schmidt-Mühlheim has affirmed that tuberculosis may pass from the peritoneum, which covers the spleen to the splenic parenchyma, and, according to Hartenstein, in the pig tuberculosis seems to be propagated from the intestine to the spleen by the lymphatic ducts; but these assertions are not exact. In cases of generalization of tuberculosis to the abdominal viscera (especially to the spleen and kidneys) and pectorals, when the lesions are old (*caseation* or *calcification*) the meat is no longer dangerous, the infectious process having long ago terminated, and the muscular tissue does not contain bacilli. In cases of *miliary tuberculosis* of the *spleen* or *kidneys*, the meat ought always to be rejected (R. Ostertag, *Zeitschrift für Fleisch und Meichhygiène*, 1891).—N. D. T.

accompanied by spasmodic spells; it is especially heard in the morning; it may be produced by making the animals rise and compelling them to walk, or making them ingest water; ordinarily it is dry; there is rarely any discharge. Breathing is accelerated and difficult; when the destruction of the lungs is far advanced it is wheezing; in the act of inspiration the ribs become lifted to excess and the nostrils are dilated. In cases of bronchiectasis, or when the pulmonary cavities are opened into the bronchi, mucopurulent or caseous matter is expectorated more or less abundantly, and the expired air has a fetid odor. The pressures exerted upon the pectoral walls or the withers frequently produce complaints and cough. In a great many patients percussion does not reveal anything abnormal; but when somewhat numerous tubercular products exist in the superficial layer of the lungs, they give, under the pleximetric blow, a dull sound which is very rarely observed upon large surfaces. On auscultation we generally hear the vesicular murmur, weakened in some regions or even entirely indistinct in the dull zones; sometimes, however, it is acute and accompanied by rattles (bronchitis); in some cases we find bronchial blowing (pulmonary cavities, bronchiectasis).

When the affection is of long standing, it is impressed upon the whole system; the hair is bristly and dull; the skin hard and dry, *sticking to the bones*; the appetite diminishes gradually, digestive troubles appear, and the lacteal secretion is diminished. There are cases where we observe a meteorization which is persistent and incurable (tuberculosis of the mediastinal ganglions with compression of the œsophagus); in others we find dysphagia and pyalism (tuberculosis of the retro-pharyngeal ganglions); sometimes we observe intermittent colics with alternating attacks of diarrhea and constipation (tuberculosis of the intestines or ganglions of the abdominal cavity), or hematuria (renal tuberculosis). We observe quite frequently hypertrophy and abnormal hardness of the explorable lymphatic ganglions (ganglions of the inter-maxillary; of the base of the ear—"lymphoma of the auricular ganglions" of the upper median and lower cervical ganglions, of the ganglions of the shoulder, elbow, the stifle, groin, mammae, etc.). In some subjects the temperature is normal; in others we observe regular remittent and intermittent fever; hyperthermia may reach—even exceed— 41° C., in evening exacerbations. The blood becomes poor, its quantity is lessened; the mucous membranes and the skin show

symptoms of anæmia (which are specially marked in the neighborhood of the mammæ and vulva), the eye is sunk in its orbit, emaciation and weakness become extreme. The animals die from persistent diarrhea or from exhaustion. The duration of the disease may vary from a few months to several years.¹

2. *Tuberculosis of the serous membranes* (pearl disease) is not marked, in the majority of cases, by any distinct manifestations. Tuberculosis of the pleura may, however, be recognized by examination of the thorax, when the serous membrane is the seat of extensive tubercular lesions, which are indicated by dulness on percussion. In some isolated cases we also observe bruits of pleural friction ("*friction of the pearls*"), which, according to our personal observations, are much more rare than is indicated by authors.

Tuberculosis of the peritoneum is betrayed by peculiar troubles of the genital organs, when they are invaded at the same time as the serous membranes. The periods of heat are prolonged and frequent; the cows, which are overexcited, bellow constantly; they jump upon their neighbors, and may be served several times without result (bulling cows). Many cows with calf abort; this accident sometimes constitutes the first symptom of *pearl disease* invading a stable (Roloff). Uterine tuberculosis is usually accompanied by vaginal discharge. The general state often remains satisfactory for a long time; but after a certain time the epiphenomena which are described in pulmonary tuberculosis appear. Finally the animals become emaciated and are overcome by cachexia.

3. *Cerebral tuberculosis* may exist as a primary disease; in the majority of cases, however, it is but a complication of pulmonary tuberculosis. Most of the time it is marked by symptoms of acute leptomeningitis. The patients are greatly excited or are subject to rabiform attacks, and convulsions and spasms similar to those present in the disease, called *spasm of the neck* (cerebro-spinal meningitis), which assume at times an epileptiform character. Later, these symptoms give place to stupefaction and to comatose and paralytic phenomena. The animals may be seen to drop abruptly ("*apoplectiform fall*"). In other cases we observe symptoms of the cerebral centres: hemiplegia, turn-sickness, hanging of

¹ We also see exacerbations which have nothing regular in their manifestations and which are produced at very different intervals. They are probably determined by external influences which pass unobserved, they are without any effective action upon healthy animals, but exercise a powerful influence upon the sick.—L. T.

the head (tuberculosis of the middle ear), paralysis of the fascia, of the oculo-motor, of the optic nerve, etc. Sometimes the phenomena of excitement appear in periodical attacks.

Tuberculosis of the spinal cord is marked by a titulating gait, by extreme flexion of the members during action, and finally by paraplegia.

4. *Tuberculosis of the mammae* may exist as a primary and unique manifestation of the process, but it is much more frequent as a secondary disease. It is characterized by a diffuse, painless, somewhat hard tumefaction which involves one or several quarters, and most commonly the posterior quarters (Bang). Contrary to what is observed in other cases of mammitis, the milk is normal in the beginning; at the end of about a month it becomes watery and contains clots, and sometimes bacilli. In many cases we have unsuccessfully looked for them in milk coming from tuberculous cows. In these animals Bollinger has found the milk virulent in 55 per cent. of the examined cases; but the pathogenic agent could only be found once in twenty. The tumefied regions of the mammae become as hard as stone. The process extends from the hind to the front quarters.

The supermammary ganglions are often of considerable size.

5. In *generalized tuberculosis* we may find all the preceding symptoms. Besides, we observe tubercular articular tumefactions (white tumors), lameness, as well as alterations of the skin and of the eyes which have been mentioned above. In very rare cases the disease pursues a subacute course and is rapidly fatal (acute miliary tuberculosis).

Bacteriological diagnosis. The diagnosis of tuberculosis, based upon the presence of the bacillus, is far from having the same importance in the cow as in man; in the former, the tuberculous expectorations being exceptional, such an examination is impossible in a majority of cases. With the object of obtaining mucus from suspicious cases, Pöls has advised the introduction of a trocar canula between two rings of the trachea; this method is rarely efficient. Injections of eserine or veratrine, which are recommended by Nocard, give us better results. In sections of tuberculous tissue it is also difficult to demonstrate the presence of bacilli; in order to find them we are often compelled to examine a large number of preparations.

In the females we may make a rapid bacteriological study of the

milk. But if this liquid sometimes contains a notable quantity of bacilli, in the majority of cases it contains spores only, which escape microscopical examination, and hence the latter loses its value as a safe diagnostic agent. In twenty tuberculous cows examined by Böllinger, eleven of which gave virulent milk, the bacteriological examination showed the bacillus in only one case.

When the diagnosis thus escapes the macroscopic technique, there is but one way left to establish it: this is the inoculation of suspected products (milk, vaginal secretions, uterine discharge, extracted lymphatic ganglions, etc.). The experiments of Arloing, Böllinger, Verneuil, and others have shown that the guinea-pig is more suited for these inoculations than the rabbit. It is sufficient to inject a small quantity of virulent matter into the abdominal cavity of this animal in order to produce a characteristic acute miliary tuberculosis.¹ At the present time the inoculation and microscopical examination are the only safe means for establishing tuberculosis during life.

The microscopical research of the tuberculous bacillus may be made in various ways:

1. By using Koch-Ehrlich's method. Spread upon a cover-glass a thin layer of the matter which is to be examined. This is dried by being passed through flame several times; then the cover-glass is placed, with the lower face downward, upon a coloring solution, the composition of which will be indicated further on, and left there from twelve to twenty-four hours. Concerning the milk, Johne has proposed to proceed in the following manner: begin by diluting the liquid in from two to five times its volume of water; then add to the mixture diluted acetic acid until the albumin is precipitated and takes the bacilli with it. A part of the deposit thus formed is placed upon the cover-glass. We have observed that we obtain finer preparations in less time when, after having placed the preparation upon its coloring bath, this is heated (heating platina and alcohol lamp) up to the boiling-point of the liquid. We allow it to become cold, and repeat the operation once or twice. The preparation of the coloring solution is very simple: Mix 5 c.c. of oil of aniline with 100 c.c. of distilled water, and filter; add 11 c.c. of an alcoholic solution of methyl-violet or of fuchsine and 10 c.c. of

¹ When we make use of only impure products (excretions, uterine discharges, etc.), it is prudent to inoculate two guinea-pigs, one in the peritoneum and the other under the skin. It is not rare to see the former die within a few days of peritonitis.—N. D. T.

pure alcohol. When the cover-glass is taken from the coloring agent it is washed with distilled water and placed for several seconds in diluted nitric acid (1 : 3), which decolors it ; afterward it is washed with plenty of water. When this operation is ended the bacilli alone remain colored. The cover-glass is placed for five minutes in a watery solution of 1 to 2 per cent. of Bismark brown or of green malachite, then it is washed again ; it is then dried and mounted in Canada balsam. The bacilli are colored dark blue or red, while all the other bacteria and the cells are of a brown or green shade. The solution must be renewed every ten or twelve days.

2. Weigert uses a 12 per cent. solution of gentian-violet, to which he adds 0.75 per cent. of liquid ammonia and 10 per cent. of pure alcohol. The preparation is placed in this solution for thirty minutes, at a temperature of 40° C. After this operation it may be left until entire decoloration in dilute nitric acid, 1 to 3, without which the bacilli lose their dark-blue coloration.

3. Gabbet has made known a new process, which is more rapid and more practical. The dried preparation is placed for two minutes in the following solution : phenicated water at 5 per cent., 100 grammes ; pure alcohol, 10 grammes ; fuchsine, 1 gramme. Immediately after it is placed for one minute in 100 grammes of sulphuric acid solution, 1 to 4, in which are dissolved 2 grammes of methyl-blue ; it is afterward washed in water, then directly examined, or it is better to wash with pure alcohol and mount it in Canada balsam. When we wish to obtain a more intense coloration of the bacilli we heat the former solution for two minutes till steam begins to appear.

Clinical diagnosis. Tuberculosis is usually marked by very vague symptoms ; thus, compared to the bacteriological diagnosis, the clinical diagnosis is very uncertain. In the beginning (in the first months) we do not possess any exact basis. Even in an advanced period of the disease the clinical symptoms permit recognition in only the minority of cases. This is a fact that some authors have wrongly contested. It has especially serious hindrances from a legal veterinary and sanitary standpoint (obligatory declaration). In a large number of cases the diagnosis can only be established by killing the subjects.¹

¹ We have endeavored to utilize Koch's lymph in order to establish the diagnosis in doubtful cases, but the reaction which it provokes does not belong exclusively to tuberculosis. In the human species authors have observed this reaction in subjects

[Dixon, who called attention to the presence of a toxic substance produced by growing colonies of tubercle bacilli, in *The Medical News* of October 19, 1889, warns us against the use of tuberculin as a diagnostic agent. After citing his clinical observations in the human economy (*Times and Register*, February 28, 1891), he concludes that cases of latent, localized tuberculosis become rapidly generalized by injections of large doses of tuberculin.—W. L. Z.]

In general the principal elements of the diagnosis *intra vitam* are the following :

1. For *pulmonary tuberculosis*: the simultaneous appearance of serious disturbances of the nutrition and phenomena indicating a pulmonary disease (cough, symptoms obtained by percussion and auscultation); hypertrophy of the lymphatic ganglions in females.

2. For *consumption*: the manifestations which give rise to the name of bullers to animals in which they are observed (nymphomania, sterility), repeated abortions, crepitation, zones of dulness, of more or less extent, and the history which is obtained. In some animals we may perceive peritoneal tubercular neoformations by making rectal exploration and in passing the hand under the last false ribs. Finally, when digestion continues normal, we must take into account obstinate chronic meteorizations.

3. In *cerebral tuberculosis*: the symptoms of a pulmonary disease,

which were affected by cancer, syphilis, cystitis, actinomycosis, etc. In animals it does not seem to furnish much more precise indications. Veterinarians who have studied the effects of tuberculin injections in bovines have obtained contradictory results. According to Guttman, Delves, Rockl and Schütz, Sticker, Bokum, Dolffs, and Nocard, these injections would constitute a good diagnostic agent. In experiments made at the veterinary school of Alfort by a commission of the Society of Practical Veterinary Medicine, and in those made recently in the slaughter-houses of Mulhausen (Temps, of September 29, 1891), we have also found in tuberculous animals constant phenomena after injections. In the hands of Kitt, Lothes, Gensert, Schwarz, Arloing, Hutyra, tuberculin has shown itself unreliable and irregular in its effects. In subjects free from tuberculosis it has determined a reaction which was distinctly marked; in others affected in various degrees it has not determined any marked disturbance. (See Arloing, Barrier: *Comptes rendus du deuxième Congrès pour l'étude de la tuberculose*, 1891, et *Recueil vét.*, 1891.) Nocard experimented upon 57 animals of the bovine species, the autopsy of which was made ultimately. All received a dose of 20 to 40 centigrammes of tuberculin; 19 from the tenth to the twenty-eighth hour showed an elevation of temperature of 1.4° to 2.9° ; in another we observed an elevation of eight-tenths of a degree. In 19 animals which reacted, 17 were tuberculous; 2 which were free from tubercles were affected, one with distomatosis, the other with ganglionic leucocythemia. In 38 subjects which showed no reaction, 2 were affected with advanced tuberculosis; 2 with acute pleuro-pneumonia; 2 with chronic pleuro-pneumonia with sequestrum, 1 verminous bronchitis; 3 pulmonary echinococcus; 1 actinomycosis of the jaw. (Nocard: *Bull. de l'Académie de Médecine*, 1891.)—N. D. T.

ganglionic tumefactions, nutritive troubles, etc., coexisting with the clinical tableau of acute encephalitis.

4. For *tuberculosis of the mammae*: a hard, diffuse, painful tumefaction of these organs, alteration of the milk, hypertrophy of the neighboring lymphatic ganglions, induration of the udder, and recognition of the bacillus.

As yet there have not been made any practical researches upon the application of the ophthalmoscope for the diagnosis of tuberculosis of the choroid and iris.

Differential diagnosis. 1. Clinically, pulmonary tuberculosis may be confounded with various chronic diseases of the lungs; such are echinococcosis and the processes which follow contagious peripneumonia. Tuberculosis of the brain may simulate simple encephalitis, cerebro-spinal meningitis, hydrophobia, malignant headache (Zundel), turn-sickness, etc. Between ganglionic tuberculosis, actinomycosis, and leukemia, we may easily be confused. A large number of observations related under the title of tubercular lymphoma of the guttural, parotid, buccal, lingual, and mammary regions, refer to actinomycosis. In some cases, besides, this is associated with tuberculosis, and sometimes the diagnosis is only established with certainty by the autopsy.

2. The morbid conditions which offer some anatomical similarity to the tuberculosis are contagious peripneumonia, actinomycosis (buccal cavity, lungs, udder, os), leukemia (liver, kidneys), amyloid degeneration (liver, kidneys, spleen), metastatic abscesses, echinococcosis (liver, lungs, spleen, kidneys), broncho-pneumonia, traumatic pneumonia, bronchitis, peribronchitis, and gastro-intestinal ulcerations.

Prognosis. The cure is possible in some cases, but only in the beginning of the affection. In general, the prognosis is very serious. The animals have to be killed.

Treatment. Any curative medication is absolutely useless. The treatment must be essentially prophylactic. The resistance of the organism has to be increased by a rational alimentation, the patients should not be used for reproduction, they have to be kept apart or slaughtered; uncooked milk must not be consumed, and the premises should be disinfected: such are the most important indications. The question of knowing if it is proper to introduce measures of sanitary police against tuberculosis, and in this relation to class this with the enzootic diseases, is yet under consider-

ation. Lydtin has advised obligatory declaration, publication, slaughtering of diseased and suspected animals, watching of the infected stables, indemnity, and penalties for violation of these measures. We doubt the necessity of these measures and the possibility of execution of some of them. An obligatory declaration is prevented by the difficulties of the diagnosis, and slaughtering of all the tuberculous animals would lead to great losses in the public treasury, admitting the general prevalence of the disease. But we consider as practical measures: 1. The slaughtering of tuberculous animals, with indemnity from the funds of a mutual insurance. 2. Slaughtering for the market descendants of animals affected by tuberculosis. 3. Destruction of the tubercular organs. 4. Boiling of the milk.

In some countries tuberculosis is considered as annulling a contract of sale. In Bavaria, Würtemberg, Baden, Hesse, a contract of sale may be annulled in *pearl disease* within 28 days, and in *pulmonary disease* (*lungensucht*) within 14 days. In Prussia the same regulations apply to tuberculosis within an interval of 8 days; in Saxony the period is 50 days; in Austria, 30 days; in Switzerland, 20 days.

MEAT INSPECTION. The identity of bovine and human tuberculosis is placed beyond doubt by numerous examples of contagion, by a similarity of anatomical alterations of these diseases, and by the existence in both of the same specific bacillus. In Germany Gerlach was the first who showed that cases of transmission to man of bovine tuberculosis are not rare. The question of consumption of meat and milk of tuberculous animals is therefore of the greatest importance from the standpoint of public hygiene.

1. The milk of tuberculous cows in an uncooked condition must be declared dangerous to man. Bang's researches show that virulent milk becomes harmless only after exposure to a temperature of 85° C., which is fatal to the bacillus. This food should only be consumed after having been boiled. In order to avoid every danger of contamination this measure ought to be generalized; milk may, indeed, contain the bacillus of tuberculosis when the cow that has furnished it shows all the appearances of perfect health. (See Bang's observations.) The use of fresh milk (which was formerly very popular as a remedy against tuberculosis of man) must be condemned in every case. It is probable that the infection may

take place by the consumption of different preparations made with tuberculous milk [cheese, whey, butter, etc. (Galtier)].

2. The meat of tuberculous animals is much less dangerous than the milk, and in some cases which have been described it may be used for consumption. In sixteen guinea-pigs which were inoculated in the peritoneum with meat-juice coming from tuberculous animals which were affected in various degrees, none contracted the disease (Bollinger). At the present time we declare the meat, as a rule, to be unfit for food: 1. In generalized tuberculosis [existence of acute miliary tubercles or chronic tubercular infiltration in two or more organs contained in various splanchnic cavities, and which are joined together, neither directly nor indirectly, nor by lymphatic vessels, nor by the vessels of the portal system, but exclusively by the channels of the main circulation (decision of the German Ministry of Agriculture, September 15, 1887)]. 2. When the meat contains tubercles. 3. When emaciation is much advanced, notwithstanding that the tubercular centres are localized; or when during life we observe the symptoms of a febrile reaction. 4. When the meat is soft and has a bad aspect.

In cases of localized tuberculosis the meat is consumed. The tubercular organs are destroyed.

The resolution which was passed by the International Veterinary Congress of Brussels (1885), recommending the rejection of all meat coming from tuberculous animals, is extreme.

II. Tuberculosis of the Pig.

GENERAL CONSIDERATION. While it is much less frequent in the pig than in the ox, tuberculosis is, however, quite common in the former species in young animals. A statistic of Lydtin, comprising all the pigs slaughtered in Baden for eight years, shows 0.02 per cent. of tuberculous subjects. At the Berlin slaughter-house during the years of 1883 and 1884 the proportion has been 0.5 per cent. Under the title of tuberculosis have been mentioned a certain number of cases in which the character of the disease remained doubtful.

Tuberculosis most frequently affects young animals during the course of the first year, contrary to what may be observed in cattle. English breeds are especially exposed to it. An artificial regimen, fattening, permanent stabling, precocity, are predisposing circum-

stances which act by weakening the constitution. The native animals are much less prone to it. Thus tuberculosis is very rare in Servia, Galicia, Bakonia, and in general in individuals which are raised and kept in pastures. The infection of young pigs is often produced by the milk of tuberculous sows; all the subjects of one litter may be affected. In some cases the germ, which is suspended in the air, penetrates into the respiratory organs. It is by this mode of contagion that the disease is sometimes propagated to all the animals of one pen. The hereditary transmission must be admitted, as whole families were found to be affected by tubercles. Numerous observations establish the fact that the pig may become infected by ingesting milk or preparations of milk coming from tuberculous bovines (Mürdel, Utz, Baumgärtl), or infected cadaveric detritus (Holten, Kolb). A contamination by expectorations of man is extremely probable, but not demonstrated.

Pathology and pathological anatomy. Tuberculosis of the pig generally begins in the digestive apparatus. According to Ostertag, we find in the intestinal grêle and the cæcum a tubercular inflammation and ulcerations of the mucous membrane, with tubercles in the muscular system, the subserous connective tissue, and the mesenteric ganglions. We frequently find tubercular amygdalitis, and sometimes a pharyngitis, which is accompanied by specific lesions of the submaxillary, retro-pharyngeal, and superior cervical ganglions. When the intestinal ulcers reach a branch of the portal system we see an embolic tuberculosis of the liver, and if the invasion of the bacilli is very abundant this organ becomes the seat of a miliary tuberculosis, which, in its turn, may lead to generalized tuberculosis. The recognition of the bacillus in the diseased organs is very difficult.

Tuberculosis of the middle and of the internal ear deserves special mention. According to Schütz, it has its origin in an infectious catarrh of the pharyngeal cavity; from there the process, ascending the Eustachian tubes, reaches the cavity of the tympanum, where it produces a tubercular osteomyelitis, periostitis, parostitis, and necrosis of the bones. It may invade the meninges, cerebellum, and the medulla oblongata; it may also be propagated to the internal auditory meatus, which is soon obstructed by a tubercular neoformation.

Primary tuberculosis of the respiratory apparatus is more rare. It assumes the character of a broncho-pneumonia of the base and

apex, with tubercular infiltration of the ganglions, and sometimes of the pleura.

The lungs present, especially in their superficial layers, numerous small miliary and submiliary granulations, which are gray and translucent, or tubercles which are of the size of a pea to that of a hazel-nut and very hard. These lesions ordinarily undergo in their centre caseous degeneration ; gradually they occasion a hard, cartilaginous consistency. Then the lungs take a spotted aspect, and their surface is often embossed. The sections which are made in their thickness show vast centres of caseous pneumonia, a proliferation of the interstitial connective tissue, chronic bronchitis and the lesions which constantly accompany it.

In cases of tuberculosis of the pleura, the subpleural lymphatic ganglions (vertebral and sternal), as well as the mediastinal ganglions may be invaded. Tubercular lesions of the trachea, larynx, and cervical ganglions are not common. Primary tuberculosis of the uterus is also rare.

In generalized tuberculosis the lungs are invaded in almost all cases (100 per cent.), the liver in 90 per cent., the spleen in 80 to 85 per cent., the buccal and the pharyngeal cavities and also the intestine in 80 per cent., the kidneys and serous membranes in 30 per cent., the bones in 15 to 20 per cent., the mammæ in 50 per cent., the testicles, uterus, and articulations in 1 per cent. (Ostertag). By this statistic we see that in the pig the spleen is more often tubercular than in the ox ; on the other hand, in this latter the serous membranes are more often affected.

For a diagnosis of local tuberculosis and general tuberculosis Ostertag gives the following fundamental rules :

I. Tuberculosis is local in the broad sense of the word.

1. When one organ and its lymphatic ganglions only are affected (intestine and mesenteric ganglions, for example).

2. In some instances where the disease affects two organs like the tonsils and intestines, as well as the submaxillary and mesenteric ganglions, or when, in cases of intestinal tuberculosis, the liver only shows isolated embolic centres.

3. In cases of tuberculosis of the lungs, of the guttural cavity, and of the intestines, when the infection has not occurred in an embolic way.

II. Tuberculosis is called general :

1. When we find generalized embolic centres, acute miliary

tuberculosis, and primary tubercular lesions of several or even of one organ only (miliary tuberculosis of the lungs with tubercular lesions of the intestines and of lymphatic ganglions which are associated with them).

2. When we recognize limited embolic centres (general chronic tuberculosis) and primary lesions in at least two organs (intestines or lungs and liver; lungs or liver and uterus, for instance).¹

SCROFULOUS OR CASEOUS ENTERITIS. The disease of the pig designated and described by Roloff is imperfectly known from an etiological standpoint. We ignore the fact as to whether or not it represents a form of tuberculosis, or whether it represents the influence of another specific affection (infectious pneumonia?). It is characterized by peculiar alterations of the intestines.

The loops of the large intestines are indurated and united to one another, resemble thick bolsters, which are separated by depressions and marked by contractions, which give to the organ a moniliform aspect. The intestinal walls are thickened and covered with button-like elevations which contain a caseous centre. Under the serous membrane and in the submucous connective tissue we find miliary nodules. The intestinal mucous membrane is sometimes covered by reddish-brown hemorrhagic centres, at other times by necrotic islands or superficial scabs, and is finally covered with caseous productions. Peyer's patches are tumefied and ulcerated. There are also found upon the mucous membrane extensive ulcerations with sharp, indurated borders disposed like bolsters. The ileo-caecal valve sometimes protrudes in the shape of a long, hard cone, with a slaty surface and ulcerated borders. We may also find cicatrices with contraction of the intestine. The hypertrophied mesenteric ganglions contain caseous centres.

Symptoms. The manifestations of tuberculosis of the pig vary with the seat of the lesions, and in many cases there is no apparent symptom to be observed.

Young pigs affected by intestinal tuberculosis have their growth stopped and become gradually emaciated. The mucous membranes become pale, the skin is covered by blackish-gray crusts (soot of young pigs). We observe various troubles of digestion: vomiting, meteorization, colics, and diarrhea. The temperature alternates from a high to a normal condition. Nutrition becomes more and more impaired; the flanks are flabby, the belly hanging, the eye

¹ See note, page 473.

sunk in the orbit. Sometimes, through the abdominal walls, we feel the intestinal loops, which are joined into a hard mass of the size of both fists and irregularly bosselated on its surface; often palpation is painful. Death generally occurs after a few months. When the lungs and intestines are affected it may take place at the end of a few weeks.

Cerebral tuberculosis is marked by a complex symptomatic apparatus. We observe automatic movements (ring action, pointing, rolling), spasms, convulsions of the muscles of the trunk and extremities, nystagmus, etc.; there are often paralysis: hemiplegia, unilateral paralysis of the tongue, inclination of the head, paralysis of the muscles of the eye, ear, eyelids, unilateral paralysis of the face, with deviation of the snout.

In pulmonary tuberculosis we observe in the beginning an abortive, dry cough, which later becomes very painful and generally occurs in exacerbation, accompanied by vomiting. The respiration is accelerated and dyspnoic; the animals lose flesh little by little. The duration of the disease varies from several weeks to a few months; in young subjects it has a more rapid course than in adult animals.

Tubercular neoformations of the external auditory meatus, tumefaction of the lymphatic ganglions of the cervical region, and intra-ocular tubercular eruption (Azary) are important diagnostic characters. The demonstration of the bacillus in the lesions is more difficult than in the ox. It is only found very rarely even in the sections.

Differential diagnosis. Tuberculosis of the pig being mostly recognized only after death, the diagnosis must especially be considered from an anatomical standpoint. We must particularly mention the similarity offered by tuberculosis with the chronic process of infectious pneumonia. Based upon the facts which were obtained in the Berlin slaughter-house, Ostertag has shown that the differential diagnosis must be based upon the following facts:

1. In tuberculosis we may find alterations in all the organs. The parenchyma contain caseous centres and non-softened tubercles. The lymphatic ganglions of the diseased organs are always affected and hypertrophied. Caseous degeneration generally follows calcification very rapidly; it is produced at the same time in numerous points in the tubercular agglomerations, and the invaded ganglions contain many small softening centres.

2. In chronic infectious pneumonia the lesions are ordinarily limited to the lungs; they are more rarely found in the intestines and the skeleton. The pulmonary lobes contain cavities and sequestra, the dimensions of which vary from that of a pea to that of a fist; there is almost always adhesive pleurisy. Among the lymphatic organs the bronchial, inguinal, and sub-glossal ganglions are most frequently affected; they have always undergone complete caseous transformation, and form round pouches filled with a concrete pus which is arranged in concentric layers. In the contents of the abscesses it is easy to find the bacteria of infectious pneumonia.

Tuberculosis and chronic infectious pneumonia are sometimes associated. In such cases the former may easily be recognized by an inoculation to the guinea-pig. As we have said, the bacteriological researches only exceptionally permit us to recognize the bacilli.

III. Tuberculosis of the Horse.

AFFECTED ANIMALS. Tuberculosis is a very rare disease in the horse. The few observations which have been mentioned are not free from criticism. On the other hand, it is likely that a certain number of clinical cases described under the name of "doubtful glanders" belong to tuberculosis.

The almost constant and often primary alterations of the lungs (Nocard, Johne, Humbert) seem to indicate that the infection is usually produced by this organ. According to Csokor, the intestinal mucous membrane is the principal seat of entrance for the bacillus. Nothing exact is known as to the source of contamination (horse, ox, man?). However, Lehnert has observed a case of contagion from the ox to the horse by simple cohabitation (in colts placed in infected stables).¹

¹ I have made several unsuccessful attempts at inoculation from the ox to the horse and from horse to horse. I killed a horse a short time ago which had been inoculated five years ago. He had shown at the point of insertion an induration which had become absorbed; at its autopsy I did not find any tubercles.—L. T.

It is difficult to infect a horse by inoculations of tubercular matter. We may, however, be successful by using large doses. Gilbert, Roger, and Cadiot have transmitted tuberculosis of the cat to the horse by intravenous injection of 4 c.c. of a dilution prepared with pulmonary tubercles. The animal died within one month, after having shown all the symptoms of double pneumonia. Both pulmonary lobes were overrun with extremely close and almost contiguous granulations, the sections of which showed characteristic bacillary accumulations. No tubercular alterations were recognized in the other organs.—N. D. T.

ANATOMICAL ALTERATIONS. They much resemble those of tuberculosis of the ox. According to the cases they are found located in one organ (lungs, pleura) and more or less generalized. In the lung we sometimes recognize miliary tubercles, at other times tubercles reaching the size of a walnut, and which have undergone caseous degeneration in their central portions; by becoming joined they may form voluminous hard masses containing centres of yellowish softening, the contents of which swarm with bacilli. The bronchial ganglions are sometimes the size of a man's fist; their consistency is hard, their surface bosselated, and their capsule thickened; on section they show a gray coloration marked by yellow spots. In their neighborhood the pleura is much thickened.

The pleural and peritoneal serous membranes frequently present alterations similar to those of the so-called consumption of the horse (*pommelière*). Sometimes the former is covered with isolated tubercles; in other cases it presents agglomerations arranged in the shape of grapes or in a thick bosselated layer. The parietal and visceral peritoneum, the epiploon, and mesentery present similar lesions. We find more or less voluminous tubercles in the liver, spleen, and kidneys; the corresponding ganglions and those of the mesentery are also affected. Tuberculosis of the bones has been observed (ribs, dorsal vertebra) with destruction of the bone tissue, formation of caseous centres, and periosteal vegetations. Finally, we may find the ordinary lesions of intestinal tuberculosis.

Symptoms. The clinical picture of tuberculosis of the horse has nothing characteristic. Generally we observe the symptoms of asthma: cough, permanent dyspnœa, emaciation, etc. In some horses emaciation appears very rapidly. We find sometimes pulmonary dulness, bronchial blowing and râles. Nocard has observed a very abundant polyuria and irregular variations of temperature (1° to 1.5° C.); in one case he noticed the peritoneal tubercular lesions on making a rectal exploration. Csokor has seen tuberculosis evolve with symptoms of serious intestinal disease.¹

¹ The main symptoms of tuberculosis of the horse are rapid shortness of breath, a more or less marked dyspnœa, emaciation, abundant polyuria; later, lessening of the appetite or complete anorexia, a staggering gait, an elevation of temperature varying from 1° to 2° ; in some cases we may hear a small dry, abortive cough without sound, and a mucous discharge which is not abundant and runs out of both nostrils; on auscultation we at times hear a hard vesicular murmur, at other times crepitation; percussion gives a normal resonance, or it may also be attenuated to some extent; we

In some of the clinical cases described under the name of *consumption of young horses* by Strans, Träger, etc., it is likely that they had to deal with tuberculosis. Descriptions of this disease correspond as well with tuberculosis of the intestines and mesenteric ganglions (tabes mesenterica, phthisis). The growth of young horses is arrested and they become emaciated; the hair is dull, brittle; the flank is hollowed and the abdomen contracted; the patients suffer from digestive troubles, slight colics, with alternations of constipation and diarrhea. They soon become very weak, and die from exhaustion induced by colliquative diarrhea.

The main lesions observed at the autopsy were: hypertrophy, induration, caseous degeneration of the mesenteric and bronchial ganglions, as well as of the catarrhal lesions of the gastro-intestinal mucous membrane. We do not possess as yet any definite facts upon the nature of this trouble.

Differential diagnosis. In some cases tuberculosis may be differentiated from glanders at the autopsy only, and by an anatomico-bacteriological examination. Recognition of the bacilli by means of Koch-Ehrlich's method insures a diagnosis. According to Csokor, the lesions of glanders and those of tuberculosis possess anatomico-histological characters sufficiently peculiar to enable us to distinguish these diseases. The pulmonary tubercles are not so distinctly delimited as the glandered nodule; they have a very marked tendency to form masses, and caseous degeneration affects them in an isolated and successive manner. In miliary tuberculosis of the lungs the lesions are formed by layers of cellular elements which are morphologically different—giant cells, epithelioid cells, round cells, while the glandered nodules are formed by caseous masses surrounded by round cells and connective-tissue fibres.

IV. Tuberculosis of Other Domestic Animals.

1. In the *dog* tuberculosis is not so rare as has been admitted up to the present time. It is probable that in a majority of cases it is of human origin: the dog contracts it by licking virulent expectorations. Several authors affirm this mode of infection. We have gathered a few personal observations which testify in favor of

may exceptionally observe hypertrophy of the pectoral ganglions. (See Nocard: Contribution à l'Étude Clinique de la Phthisie Tuberculeuse chez le Cheval, au Receuil Vét., 1885.)—N. D. T.

this theory: in one case three dogs belonging to the same owner became contaminated and died of generalized tuberculosis.

Mostly the symptoms are those of chronic pulmonary disease with considerable emaciation (pulmonary phthisis). In two patients we have been able to recognize the bacillus in the discharge by the method of Koch-Ehrlich. However, the *intra-vitam* diagnosis is difficult in the dog. By microscopical examination we may differentiate tuberculosis from chronic pneumonia (consecutive to disease of young age), from pulmonary carcinomatosis and sarcomatosis. In one patient we obtained a striking improvement with prolonged inhalations of creoline ($\frac{1}{2}$ per cent.).

At the anatomical examination we find in the lungs one or several centres of tubercular pneumonia and miliary tubercles; there are also lesions of the bronchial ganglions and tubercular pleurisy.

In intestinal tuberculosis the mesenteric ganglions and the other organs of the abdominal cavity (peritoneum, liver, spleen, etc.) are affected.

2. In the *cat* Bollinger has described two cases of miliary tuberculosis with invasion of the pancreas. In another observation mentioned by Zschokke this author suspected that the animal was infected from a human source.

3. The cases of tuberculosis observed in *sheep* and *goats* offered a great anatomo-pathological resemblance with tuberculosis of the ox. In one of these Lydtin ascribes the contamination to tubercular oxen. As main symptoms we have mentioned cough, emaciation, and anæmia.

Tuberculosis affects a large number of wild foreign animals living in captivity (monkey, lion, tiger, etc.). It is especially to this that we must ascribe the shortness of life in these animals.

V. Tuberculosis of Poultry.¹

AFFECTED BIRDS. Ornithic tuberculosis is one of the most frequent diseases of the bird species. In 600 autopsies in chickens made by Zürn 62 died from tuberculosis (10 per cent.). It affects

¹ The nature of tuberculosis of birds is yet in discussion. The lesions which are produced by it contain bacilli which are similar to those of human tuberculosis and possess the same histochemic characters. Bollinger, Nocard, Lemallerée, Guérin, and Dixon have mentioned facts tending to establish the identity of these two affections. Soon after Strauss and Wurtz announced that they had failed to transmit tuberculosis by making six hens and one cock ingest virulent expectorations for one year. Rivolta

all breeds (chicken, pigeon, pheasant, peacock, etc.), and it exists frequently in an epizootic state. The intestine is the seat of specific lesions in a large number of cases; the infection takes place, as a

and Maffuci recognized that the bacillus of ornithic tuberculosis possesses only slight pathogenic properties in mammiferous animals. Finally, at the Medical Congress of Berlin, Koch stated that he was no longer able to assimilate entirely tuberculosis of chickens to that of man.

Gilbert, Roger, and Cadiot have taken up this question. They have recognized that tuberculosis of gallinacæ is transmissible to chickens; that the rabbit contracts tuberculosis easily and dies in two to three months from generalized infection; that the guinea-pig, which is less resistant than the rabbit to human tuberculosis, is much more so than this latter animal to ornithic tuberculosis, and that it is exceptional, by inoculation, to provoke a general infection in this animal; according to the cases the subjects remain unaffected, or they show at the point of inoculation a lesion which disappears in the course of time, or, but much more rarely, a discrete visceral infection is produced which has a tendency to heal.

These authors have also established that ornithic tuberculosis differs from human tuberculosis by its histological characters: "In the chicken the lesion is mainly composed of a vitreous mass, which is surrounded by a border of special epithelial cells; these are arranged perpendicular to the vitreous zone and provided with multiple nuclei, principally agminated to the cellular extremity which is most distant from this zone. In the pheasant the aspect is entirely different; it is an accumulation of epithelioid cells which undergo molecular retrogression in the central part of the neoplasm; around these a connective-tissue ring is formed which becomes infiltrated with amyloid matter. The aspect is so different in these two neighboring animal species that we might believe in having to deal with two distinct affections; but this is not the case; in fact, a tuberculosis of the pheasant, if inoculated in the chicken, produces in this bird the same lesions which are commonly observed."

Strauss and Gamaleïa have not succeeded in transmitting human tuberculosis to the gallinaceous species by subcutaneous, intra-muscular, and intra-peritoneal inoculations. Having observed besides that the characters of the cultures of human virus and ornithic virus are very different, and that the dog, which contracts human tuberculosis easily, resists almost completely the tuberculosis of birds, they have concluded that the human bacilli and the bacilli found in birds constitute two different microbic species.

Gilbert, Roger, and Cadiot have succeeded in transmitting human tuberculosis to chickens; in forty fowls which had been used for experimental purposes, three which had been inoculated at the same time into the veins and peritoneum showed at the autopsy lesions of generalized tuberculosis. They consider the human and poultry tubercular bacilli as being derived from one source, and are advocates of the *uniformity* of tuberculosis of mammifera and poultry.

The results which have been obtained by these investigators have been confirmed by Courmont and Dor, who have also succeeded in transmitting tuberculosis of mammifera to chickens by operating through the digestive passages and the subcutaneous connective tissue. Here are the conclusions which they have drawn from their researches:

1. The chicken is not absolutely refractory to human tuberculosis.
2. The digestive tract is very inferior as a mode of entrance for the affection than is the subcutaneous procedure.
3. The bacilli of human tuberculosis may in rare cases be propagated in series in the chicken by subcutaneous inoculations.
4. The bacillus of human tuberculosis and that of tuberculosis of birds do not constitute two distinct species, but rather two classes of the same species.—N. D. T.

rule, through the digestive apparatus. The excrements of diseased birds often contain a large number of bacilli; it is by them that contamination is usually produced. But the expectorations of man (especially in hospitals) may also become the contagious agents. The same is the case with milk or meat of tubercular bovines. In poultry establishments the infection is considered as *hereditary*.

[Dixon affirms that bovine, human, and gallinaceous tubercle bacilli belong to the same species.—W. L. Z.]

Pathological anatomy. The alterations are mainly found in the liver, spleen, and intestines. The liver shows lesions which are of variable aspect, either of miliary granulations or tubercles which may reach the dimensions of a hazelnut or a walnut. They are isolated or agglomerated, and offer, according to their age, a white, gray, or yellow coloration. Their central part has undergone caseous degeneration or calcification. They contain a large number of bacilli. Similar alterations are found in the spleen. In the intestinal mucous membrane we frequently observe tubercles of the size of a millet-seed or that of a pea, and which become ulcerated in course of time. They are also found in the gastro-intestinal serous membrane and other regions of the peritoneum, in the mesentery, the kidneys, ovaries, etc. Tuberculosis of the lungs, of the air-pouches, heart, and pericardium are more rare.

The lymphatic ganglions, articulations (of legs and wings), and the periarticular tissues are often the seat of considerable tubercular lesions (tumefactions and abscesses with caseous contents; tubercular arthritis and periartthritis). They are also found in the bones, the skin, and the subcutaneous connective tissue (tubercles and ulcerations).

Symptoms. The symptoms of tuberculosis of poultry have nothing very significant. The patients are weak; they become gradually emaciated, sometimes like skeletons, chronic disturbances of digestion are observed (inappetence, vomiting, diarrhea); the comb becomes flabby and pale, the conjunctiva and buccal mucous membrane are discolored. At an advanced period we may observe paralytic symptoms. The diagnosis is relatively easy when tuberculosis becomes localized in the joints, bones, or skin, in the shape of articular or bony tumefactions, tumors, or bad-looking cutaneous ulcerations. The course of the disease is generally quite slow.

ACTINOMYCOSIS.

Etiology. Actinomycosis is determined by a cauliflower-like fungus—the *Actinomyces bovis*—which seems to belong to the fungi group. It has been seen in the ox, pig, horse, and elephant (Burke). It has also been found in man. *Actinomyces* was observed by Peroncito and Rivolta in the years following 1860, and by Hahn in 1870; but the first exact description of this parasite was given by Bollinger in 1877. After having studied it from a botanical point of view, Harz gave to it the name of *Actinomyces* (rayed fungus). When examined with the naked eye it has the aspect of a spheroidal granulation the size of a particle of sand, and has a coloration varying between white and sulphur-yellow; its diameter is 0.1 mm. at least. At the microscopical examination it offers a ray-shaped arrangement, which reminds us much of the crystalline forms of fatty acids; it is formed by a series of long cells which are club-shaped, and the extremity of which is spheroidal or pyriform (conidia), and corresponds to the periphery of the granulation; in the centre of this we observe a very fine mycelium with intricate filaments (hyphes); the conidies become segmented by gemmiparity or by scissiparity. These fungi often undergo calcification. The first inoculations in the calf and ox were made by Johnne.

The actinomycetes may penetrate the organism in different ways, but it is usually through the digestive apparatus that they gain entrance into the system (buccal cavity, stomach, intestines). Plants are their favorite location. According to Brazzola, they vegetate principally on the *hordeum murinum*. Upon fragments of this grass which had penetrated into the gums this author has found between the vegetable fibres accumulations of actinomycosis. Johnne and Piana have found the fungus upon particles of grain lodged in the tonsils and the tongue of the ox. It is generally admitted that it becomes first implanted in small wounds of the buccal mucous membrane, the excretory canals of the glands, and in the alveoli of diseased teeth. Once introduced into the tissues it continues to grow and invades the neighboring parts. The infection may also take place in a pulmonary way through wounds of the skin and the orifices of the teats. Lastly, it seems that a generalization by the circulation may be produced as in tuberculosis¹

¹ Since Johnne has succeeded in transmitting actinomycosis to subjects of the bovine species, other investigators have made upon other animals numerous attempts at in-

Frequency. In 105 cases of actinomycosis of the ox observed by Claus the maxillæ (notably the lower) were affected in 51 per cent. of the cases; the tongue in 29 per cent.; the pharyngeal cavity in 7 per cent.; the larynx and trachea in 6 per cent.; the lungs, abdominal viscera, and the cranial bones were only exceptionally invaded. According to Immenger, the affection exists mainly in the Palatinate and Franconia. In Bavarian Palatine this author finds yearly more than 100 cases of actinomycosis of the ox. He has observed that the disease is particularly frequent in young animals at the period of dental eruption; it is ordinarily located upon the head and the neck (85 to 90 per cent.), more rarely upon the tongue (4 to 8 per cent.). The actinomycoma of the base of the tongue starts generally from the non-perforated openings of Morgagni.

Peletti, who has observed actinomycosis in hundreds of cases, noticed that it is especially common after aphthous epizootics; he concludes from that fact that the ulcerations of the buccal membrane play a rôle in its pathology. The tongue was affected in most cases. In Denmark actinomycosis is most frequently developed upon the soft parts of the face and neck (Bang); in England it is generally found on the tongue.

We give below the figures furnished by the statistics of slaughter-houses concerning the frequency of actinomycosis. In 100,000 oxen killed in Berlin in 1885-86 there were 21 cases (1 per 5000), and in 300,000 pigs 2 cases (1 per 150,000); in Augsburg, in 23,000 oxen 8 cases (1 per 3000); in Bremen, in 8500 oxen 2 cases (1 per 4250), and in 25,000 pigs 3 cases (1 per 8000); in Stuttgart, in

oculation which have given some positive results in rabbits and guinea-pigs. Hammond is said to have observed one case in the sheep, and Vachetta another in the dog. They have succeeded in cultivating the actinomycetes in various mediums: in solidified serum (Israel), agar in the shape of glycerin (Kischensky), in gelatin, potatoes, egg, liquid serum, milk, and broth. The most favorable temperature for their development is between 32° and 37° C; their vegetation is retarded at 40° to 41°, and stops at 52°; the culture is killed in ten minutes by a temperature of zero. Liebman has recognized that the activity and vegetability of actinomycetes become attenuated in the animal organism, but the parasite regains its vegetative strength and pathogenic power when it is made to grow upon a vegetable. By inoculating it in grain it multiplies at the same time as the latter is developed, and invades the whole plant (Liebman). These facts clear up the etiology of the affection; the difference in the regimen explains the frequency of actinomycosis in herbivora and its rarity in carnivora. (See Roger: *art. Actinomycose, Traité de Médecine*, Paris, 1891.)—N. D. T.

12,000 oxen 12 cases (1 per 1000); in Hanover, in 10,000 oxen 1 case (1 per 10,000).¹

Anatomical alterations. These present a certain analogy to the lesions of tuberculosis. Around the point where the fungi aggregate the inflammatory reaction first produces a granular mass, which is soon accompanied either by neoformations of tubercular appearance or by rounded or lobulated tumors which are of variable size. In general these lesions are very soft; they have a sarcomatous aspect and a reddish-yellow color; at times they have the density of fibroma, with a whitish-gray coloration; in other cases they are of spongy consistency. They are formed of a connective-tissue stroma, in which are enclosed numerous nodules of various dimensions from a millet-seed to a pea; often they are scarcely visible, present the histological constitution of granular tumors, and contain actinomycotic particles which are sulphur-yellow in color; they may become confluent and form small, tuberculiform masses. When the actinomycoma undergoes purulent destruction, we see in its centre the development of a cold abscess which is more or less extensive and surrounded by a soft, granular tissue which also contains actinomycetes.

Actinomycosis of the *ox* is generally localized upon the maxillary bones, where it produces considerable tumefactions and swellings, which were formerly designated as *spina ventosa*, *sarcoma of the jaw*, *farcy (worm)*, etc. On the tongue we notice prominent tubercular masses of the size of a nut, and some ulcerations; by connective-tissue neoformation the organ may acquire enormous dimensions and great toughness, from which the name "wooden tongue" has been derived. The pharynx, œsophagus, and larynx are sometimes obstructed by polypus or sessile tumors called formerly "lymphoma of the pharynx," or "tumors of the œsophagus." These alterations seem to exist in the neighborhood of the larynx and parotid gland ("the parotid lymphoma," "ganglionic tubercles," "scrofu-

¹ Actinomycosis is frequent in Russia. In two years Ivanow, Inspector-in-chief of the Moscow slaughter-houses, observed 2000 cases. It is less common in the United States than has been stated. The statistical report of the slaughter-house inspectors show but a proportion of 2 per 1000 in animals of the bovine species (Salmon). In cattle imported from Canada to England the proportion reached 2 per cent. (Barrett). In France this disease is quite rare. It was first seen by Nocard in 1884. Since then a number of instances have been reported. In 1890 we recognized a case in a cow brought to the Alfort school for examination by a breeder of the neighborhood.—

lous blotches," "hedge-hog," etc.). They have been found in the stomach and intestinal and mesenteric ganglions. When the lungs contain a large number, they may simulate the lesions of miliary tuberculosis; the bronchial ganglions are invaded. In some cases they are found under the skin ("chronic tubercles"), in the bones (articulations, cervical vertebra), in the muscles, the peritoneum, etc. Actinomycosis, like tuberculosis, may become generalized.

In the *pig*, actinomycomas have been found in the lower jaw, larynx, lungs, mammae, and bones. The invaded tissues form abscesses, they become hollowed by fistulas, and allow the discharge of a purulent matter containing numerous actinomycetes. Actinomycosis has been seen in one instance by Perroncito in the bones of the *horse*, and in another by Zschokke in the tongue.

ACTINOMYCES MUSCULORUM SUIS. In the muscles of the pig, Duncker has found another fungus which also has conidies which have the shape of clubs and are arranged in divergent rays. Duncker and Hertwig have given to it the name of *Actinomyces musculorum suis*, in order to distinguish it from *Actinomyces bovis*. It is mainly located in the muscles.

This actinomyces has been found in 187 pigs. When enlarged 40 or 50 diameters it appears as a round corpuscle which is distinctly outlined and of brownish-gray color. An enlargement of 500 diameters has permitted us to discover particles which are located between filaments enlarged in their free extremity. The muscular substance of the neighborhood is altered; the muscles are infiltrated, softened; the meat has often a disgusting look, which alone should cause its rejection for consumption. According to Hertwig, infection generally takes place in summer or in the beginning of autumn. This parasite has not the characteristic uniform aspect of the *actinomyces bovis*. Moreover, in that affection the meat is only altered at an advanced period of mycoses (Johne).

BOTRYOMYCOSIS OF THE HORSE. In the spermatic cord of castrated horses Rivolta found fungi which he described under the name of "Discomyces equi," and which he has related to the *actinomyces*. Rahe has given them the name *micrococcus botriogenus*, and Johné that of *micrococcus ascoformans*. They produce a connective-tissue neoformation and induration of the testicular cord (mycofibroma or mycodesmoids). These parasites are micrococci, and not fungi. Böllinger, who has found them in multiple fibromatous tumors of the horse, has advised the designation of the

disease by the expression botriomycosis (colonies in the shape of grapes).

Symptoms. Actinomycosis is especially of great interest from the surgical and anatomo-pathological standpoints. Sometimes, however, it determines symptoms which enter into the domain of internal pathology. When the tongue is affected prehension and mastication are hindered; the organ is tumefied and painful to the touch; there is often abundant ptyalism. Actinomycosis of the pharynx provokes dysphagia, and that of the larynx dyspnœa. If the neck is invaded, we find the parotid region distended and covered with more or less extensive tumors. Actinomycosis of the cervical vertebra may lead to spinal paralysis by compression and atrophy of the spinal cord. That of the lungs determines the symptomatic appearance of chronic pulmonary disease. The course of the disease is also very slow. As in tuberculosis, we observe cases of spontaneous cures by encysting (Turminger).

Differential diagnosis. We may confound actinomycosis with tuberculosis (pulmonary and ganglionic), pleuro-pneumonia, and apthous fever. The diagnosis is made by recognizing the actinomycomas. Bang has insisted upon the tendency possessed by the actinomycomas to find an exit. In order to search for the fungus double staining with orseillin and gentian-violet has been used (Weigert), or orceine (Israel). Quite recently Baranski has used picrocarmin. After having spread a very thin layer of pus upon a lamella we dry the former in the open air and pass it through the flame of an alcohol lamp; the preparation is then colored with picrocarmin (two to three minutes of contact), washed in water or alcohol and placed in water, glycerin, or Canada balsam. The actinomycetes show a beautiful yellow coloration, while the rest of the preparation is colored red.

Csokor has recommended double staining with picrocarmin, and Zschokke the use of hematoxylin or eosin.¹

Treatment. This is mainly surgical and consists of extirpation or cauterization of the actinomycotic tumors. The use of injections of iodine, sulphate of copper, phenic acid, etc., has been advised to

¹ Crookshank has found that actinomycetes of the ox do not behave in the same manner as those of man toward some staining agents. In the former the rods become colored very distinctly and the central portion of the nuclei remains colorless; in the latter they resist the stain, while the central portion appears as colored, intermixed filaments. The author admits, nevertheless, the identity of the affection in man and in cattle.—N. D. T.

be made in the altered tissues. Türthmeyer has recommended painting the tongue with tincture of iodine and the administration of ioduret of potassium (10 grammes per day in $\frac{1}{4}$ of a litre of lukewarm water); this treatment, if continued for twelve days, has several times given him good results; in seven patients he obtained cure. Bass has gathered observations which confirm the efficiency of these agents.¹

ACTINOMYCOSIS OF MAN appears ordinarily in the form of suppurating phlegmons or abscesses. In most patients the bones of the face are affected; metastasis may take place in the lungs, liver, kidneys, brain, etc. The infection very likely takes place as in animals, by the intermediation of vegetables which are covered with fungi; it is possible also that man may be contaminated by diseased oxen.² The meat of animals affected with actinomycosis should be rejected for consumption.

GLANDERS.

HISTORY. Glanders is one of the equine diseases which has been longest known. Farcy and glanders of the donkey are mentioned in the writings of Aristotle and Hippocrates. Among the Greek and Roman authors, Apsyrte and Végecè have described glanders (*malleus*); Végecè distinguished several forms, especially nasal glanders and the cutaneous form (*malleus humidus*, *farciminosus*). In the middle ages German law considered it a disease setting aside a sale. It was then designated by very different expressions (principal disease, deathly disease, *profluvium atticum*, etc.). From the seventeenth century its contagiousness has been known: Solleysel (1664) thought that its transmission was brought about through the air; Van Helmont (1682) considered it as identical with syphilis of man. During the eighteenth century Gaspard de Saunier (1734) gave directions concerning disinfection of the stables;

¹ For parotid actinomycosis Harms has recommended frictions with an ointment composed of equal parts of arsenic and lead (*Deutsche Zeitschrift für Tiermedizin*, 1888).—N. D. T.

² Facts have been gathered tending to establishing contagion of actinomycosis of animals, or even from man to man. Israel has mentioned a case of a coachman who, having drank of water from the same pail used for his horse, which was affected by actinomycosis, contracted the disease; Baracz mentions the history of another groom who infected the bride by kissing her upon the mouth. In several cases men have become infected in handling diseased animals or in making their autopsy (Roger, *loc. cit.*)—N. D. T.

Garsault (1741) and Bourgelat (1764) advised the slaughtering of glandered horses and isolation of suspected animals. The identity of glanders and farcy had already been admitted at that period.

Toward the end of the last century two veterinarians—Abildgaard and Viborg—demonstrated by numerous experiments the transmissibility of glanders. These authors recognized that the germ of glanders is fixed and "*volatile*;" that transmission of the blood of the patients gives fewer positive results than inoculation of secretions and pus; finally, that virus dried or heated at 45° C. lost its virulent properties. Similar experiments made in England by Coleman and Delabere-Blaine established the fact that the discharge is particularly virulent.

In the beginning of this century the contagiousness of glanders was doubted. In France the Alfort School defended the theory of spontaneity of the Lyons School, where the old doctrine was taught. The doctrines of Alfort prevailed, the measures of sanitary police were repealed, and glanders spread to a disastrous extent in the whole French territory. Some (Dupuy) considered it as having a tuberculous character; others (Vatel, Bouley, etc.) looked upon it as simple pyemia. Rayer (1837) recognized the transmissibility of glanders to man, and when Chauveau (1868) had given proof that the virulence was inherent to the solid particles of the putrid fluids, the theory of contagion was again established.

In Germany, also, toward the middle of the present century, glanders was considered as a spontaneous disease. The theory was that it could be produced by injecting pus into the blood, and it was generally admitted that strangles underwent alterations of glanders (Herin, Funke, etc.), and this was considered as a tubercular disease, a pyemia, diphtheria, a dyscrasia or cachexia. Virchow, Sr., asserted that glanderous tubercles are true anatomical neoformations which are autonomous, and he placed them in the category of granular tumors. Gerlach defended the doctrine of contagiousness and published important clinical works upon this subject.

Leisering was the first to give an exact anatomical description of this disease.

The bacteriological researches upon glanders date from 1868; they were begun by Zürn and Hallier. These authors found in the lesions of glanders a fungus which seemed to them identical with that which is found in the syphilitic lesions of man; they considered it as the essential infectious agent. At the same period

Christot and Kiener also thought that they recognized the bacillus of glanders; they cultivated and inoculated it in healthy animals. The researches of these authors have demonstrated that glanders is a specific infectious disease produced by a micro-organism, the properties of which they have described.¹

Bacteriology. According to Löffler and Schütz, the bacilli of glanders appear in the shape of little rods of a length varying from one-third to two-thirds of the diameter of a red corpuscle and of a thickness from five to eight times less. They are rectilinear or slightly incurved, rounded at their extremities, a little shorter and thicker than Koch's bacillus, and are frequently found together by twos and placed end to end. They may easily be colored with methylene-blue.² In gelatin they form small, limpid, yellowish drops, which later have a milky aspect. The cultures upon potatoes are characteristic; from the third day they constitute a uniform transparent layer, which has the color of bitumen; from the sixth to the seventh day they have a reddish coloration similar to peroxide of copper; toward their periphery they have a greenish shade. The most favorable medium for the culture of the bacillus is coagulated serum of the horse or sheep, or neutralized broth of the horse, ox, chicken; also the same preparation with human flesh, and, lastly, with the potato. It reaches its maximum development from 20° to 45° C.; warm weather favors its growth. Temperature below 20° C. and higher than 45° hinders its development or destroys it. Thus it is possible that it does not multiply outside of the organism (exogenous development). Löffler has not succeeded in cultivating it in decoctions of hay and straw, nor upon horse manure. Glanders seems, therefore, to be a non-miasmatic contagious disease. This bacillus is killed in one week by desiccation. In his experiments Löffler has established, however, that in

¹ In 1881-82 Bouchard, Capitan, and Charrin succeeded in isolating and cultivating the microbe of glanders in liquid mediums. With the fifth and eleventh cultures they produced glanders in guinea-pigs and donkeys. They have not defined the morphological characters of the microbe, because in cultures which were equally virulent they found it sometimes in the form of micrococci; in other instances in the shape of bacilli; cultures preserved in sealed tubes and kept from air and light could later be cultivated upon solid mediums (potatoes, agar, gelatin), and the microbe presented all the characters attributed by Schütz and Löffler to the bacillus of glanders (December, 1884), including the chocolate coloration of the potato cultures (Ch. Bouchard: *Thérapeutique des maladies infectieuses*, Paris, 1889).

² The bacillus of glanders is aërobic and mobile; it takes neither the Gram nor the Weigert stain.—N. D. T.

dried putrid animal fluid it may preserve its virulence for three months. According to Cadeac and Mabt, desiccation destroys it only when performed slowly. It resists putrefaction from fourteen to twenty-four days; in water it remains virulent for fifteen to twenty days. In moist matter it does not live longer than four months; Löffler considers this length of time as the maximum duration of virulence of the infectious matter. According to him, the statements of the reappearance of glanders by the action of virulent matters which have been preserved in stables from which the disease had disappeared for a long time—several years—are erroneous.¹ The slight resistance of the bacillus to destructive agents has induced the belief that it is asporogenic (Löffler). However, in treating old cultures upon potatoes by the process of staining spores devised by Neisser, Rosenthal has obtained forms similar to those of sporulated bacteria. We must therefore admit the possibility of sporulation.

The bacilli are killed in ten minutes by a temperature of 55° C. in five minutes by a temperature of 80° C., and by phenicated water at 3 to 5 per cent.; in two minutes by chlorine water, by 1 per cent. solution of permanganate of potassium, and by a solution of sublimate at 1 per 5000. In practice we may use as disinfectants the solution of sublimate at 1 per 1000, 5 per cent. phenicated water, and boiling water. The development of bacilli outside of the organism is largely arrested by the action of iodoform; if exposed for a sufficiently long time to the influence of this agent, they lose their virulence. In the organism, however, iodoform has but a slight influence upon them.²

¹ It is indeed almost certain that the disease is only kept latent in animals where it passes unobserved for a long time. In a stable where it existed for three years I ordered the killing of an old horse which showed but a slight mucous discharge and no enlarged gland, and this was done for the only reason that his neighbors had become affected with glanders. At the autopsy I found the lungs filled with tubercles, some of which were of long standing. Since then no other case of glanders has occurred. This fact shows how difficult it is to ascribe the infection to the premises.

—L. T.

² The activity of the bacillus of glanders may be modified by various influences. In cultures arranged in series upon artificial mediums the virulence diminishes gradually. Bouchard, Capitan, and Charrin have observed that it finally becomes attenuated to such a degree that inoculation often produces but a local lesion. Gama-leia has recognized that the organism of the spermophile increases the virulence by inoculations which were made in series upon that animal the bacillus of glanders acquires such a virulence that it kills it in forty-eight hours. At the autopsy we find an increase in the size of the spleen and numerous bacilli in the blood. (Roger: *Traité de Médecine*.)—N. D. T.

THE PROCESS OF STAINING THE BACILLUS OF GLANDERS varies according to the preparation, which may be either in sections or on cover-glasses. Sections from specimens hardened in alcohol are placed for four minutes in a liquid composed of 2 cubic centimetres of a concentrated solution of methylene-blue and 100 cubic centimetres of a watery solution of potassium (1 per 10,000); then they are dipped for a few seconds into a 1 per cent. solution of acetic acid, dehydrated with alcohol, and placed in oil of cedar.

Preparations upon cover-glasses are dried as usual, and placed for five minutes in a bath composed of equal parts of aniline water, a concentrated solution of gentian-violet, and a solution of potassium (1 per 10,000); they are then dipped for one second in a 1 per cent. solution of acetic acid, having previously been colored in yellow Rhine wine made so by the addition of a solution of tropeolin; and finally rapidly washed in distilled water.

AFFECTED ANIMALS. This disease is almost confined to the *equine* race, and is found in the horse, donkey, mule, and hinny. But by natural or experimental infection it may also be developed in other species and man. Considered from the standpoint of their receptivity to the glanderous virus these species are to be classified in the following order: field-mouse, guinea-pig, cat (lion, tiger), dog, goat, and rabbit; then come the sheep, pigs, and pigeons; the two latter subjects are very slightly sensitive to it. The ox seems to possess complete immunity. Glanders has never been observed in this animal, and glandered horses have often been isolated by placing them in stables with animals of the bovine species. Up to the present time the common mouse, the white mouse, the rat, chicken, and chaffinch have proved refractory to glanders.

[A personal communication from Dr. Francis Bridge, Veterinary Inspector for the Pennsylvania State Board of Agriculture, contains the following:

“On December 13, 1886, Dr. James Clark, of Peach Bottom, Lancaster County, Pa., told me that he thought he had a very bad case of glanders in his locality, and would like me to see it. The doctor and I went to the place, which was owned by William Johnson, a laborer. The stable was dilapidated and dirty; in the place were found a horse and cow, also chickens and a pig. I found the horse to be in the last stage of glanders and farcy. The near hind leg was enormously swollen; farcy buds in various stages of development, both on the neck and under the belly; a thick, glu-

tinuous discharge from both nostrils adhered to the margin of the nose; the submaxillary and thyroid glands were much swollen; the Schneiderian mucous membrane was studded with chancreous ulcers of various sizes. The owner said that the horse had a discharge from the nose when he bought it, more than twelve months previous; but thought nothing of it, as he was told when he got it that it had catarrh. The cow stood in the stall facing the horse. This animal would hang his head over the cow-stall, and she would lick the discharge from his nose. Upon examination of the cow I found that she had a characteristic glanderous discharge from one nostril; thyroid and submaxillary glands were much swollen and very sensitive to the touch; the off hind leg enlarged and tender; the margin of the nose excoriated. I ordered both animals destroyed, and requested Dr. Clark to make an autopsy, especially on the cow, which he promised to do the next day; but the owner refused to permit the animals to be posted on the premises, so they were buried without an examination of their internal organs."

It is to be regretted that a post-mortem examination was not made in this most unique case.—W. L. Z.]

While it is to-day far less frequent than formerly, on account of the strict application of sanitary measures, glanders nevertheless remains the most dangerous of all diseases of the horse. It is found in all countries and all climates. The charts furnished by the German sanitary service during the year 1887 show that it makes special inroads in the border countries of occidental Prussia, in the provinces of Posen and Silesia, between the districts of Strassburg (Western Prussia) and Wreschen, upon the right bank of the Warthe and all along the Russian frontier toward the southwest. The principal centres of this disease are the highlands of Dantzig, Inowrazlow, the cities of Posen, Liebnitz, and a small district in Silesia (Zabrze). In central Germany it exists in a somewhat extended zone at Zellerfeld. In southern Germany we find numerous centres of the trouble, mainly in Bavaria and Würtemberg (Mellrichstadt, Hassfurth, Bayreuth, Rehan, Rodach, Waldmünchen, Schorndorf).

According to a statement of Felisch concerning the cases of glanders reported in Prussia from 1876 to 1886, the losses amounted to 20,566 horses having died or been killed. The figures for these different years are: 2740, 2848, 1994, 2182, 2357, 2297, 1568, 1879, 1525, 1176. We see that for that period the losses have

diminished more than one-half. These results were obtained by means of the enforcement of the law regarding infectious diseases. The greatest proportion has been observed in the Duchy of Posen (2.2 per cent. of the total number), western Prussia (1.5 per 1000), and Silesia (1.3 per 1000). The disease was especially widespread in the districts of Oppeln, Bromberg, Posen, Marienwerder, and Dantzic (Russian-Polish frontier). Twenty-two per cent. of the horses affected by glanders belonged to carters. Twenty cases of transmission to man (three of which were veterinarians) have been reported. The total sum of indemnity allowed was 5,630,000 francs.

A statistic of Krabbe, embracing sixteen years (1857 to 1873), gives the following figures as the annual average of cases of glanders for 100,000 horses: Norway, 6; Denmark, 8.5; Great Britain, 14; Sweden, 57;¹ Würtemberg, 77; Prussia, 78; Servia, 95; Belgium, 138; France, 1130; Algiers, 1548.² The question is as yet undecided whether this increase of glandered horses from the north to the south is due to the influence of the climate, to the higher temperature (hypothesis admitted by Löffler and based upon the fact that the glanderous bacillus possesses its greatest activity at temperatures above 20° C.), or to a more or less strict enforcement of sanitary measures in various countries. All horses are not equally sensitive to the glanderous virus. As predisposing causes we may mention: excessive work, want of care, damp and badly-ventilated stables, colds, and debilitating diseases. Thus the largest number of cases of glanders are seen in times of war.

Pathology. The contamination of a healthy animal by a horse affected with glanders may take place directly or by various inter-

¹ In a note addressed to us by Prof. Lindquist, Director of the Veterinary School of Stockholm, this figure is corrected with the remark that in the old official reports several cases of lymphangitis appear under the name of farcy which were not at all of a glanderous nature. This form of lymphangitis at the present day has completely disappeared from Sweden. In the ten years from 1877 to 1886 there were observed 199 cases of glanders, which were thus distributed: 1877, 32 cases; 1878, 31; 1879, 37; 1880, 27; 1881, 17; 1882, 29; 1883, 7; 1884, 9; 1885, 7; 1886, 3. During this period the proportion of cases of glanders was 3.9 per 100,000 horses.—N. D. T.

² In France, since the promulgation of the law of sanitary police, the losses which were caused by glanders have much diminished. In 1887 this disease was noticed in 1233 horses, representing a value of 423,000 francs. It was observed in all departments except Ardennes, Hautes-Alps, and Pas de Calais (Tisseraud: Rapport sur le Service des Epizooties en 1887). This same year the equine population was 2,908,500 subjects (Annuaire Statistique de la France). The proportion of cases of glanders has thus fallen to 42 per 100,000 horses.—N. D. T.

mediaries (harness, blankets, pails, food, litter, and man, etc.). The product of secretions of farcy ulcers and the nasal discharge are the most virulent matters, but the glanderous bacillus exists also in the organs, and the blood becomes virulent when general infection takes place. According to Cadeac and Mallet, inoculations made with blood coming from glandered horses have sixteen times given positive results, and thirty-five times they were negative.

In a majority of cases (about nine-tenths) the bacilli penetrate through the respiratory organs. When in suspension in the air they reach first the nasal mucous membrane. Their introduction into the depth of the respiratory apparatus is favored by certain circumstances; when, for instance, the animals smell at one another or the respiration becomes accelerated and deep under the influence of exertions induced by pulling. They may be carried directly into the lungs with the inspired air, and there produce small broncho-pneumonic centres.¹ Pulmonary glanders may be primary just as well as nasal glanders. In a large number of cases the lungs alone are affected, or it is upon them that we observe the oldest lesions. It is the same as cases of tuberculosis and actinomycosis. Most authors have admitted the possibility of primary pulmonary disease in glanderous infection (Leisering, Gerlach, Böllinger, Roloff, Pflug, Rabe, Röhl, Csokor, Johné). However, we must point out that certain clinical facts mentioned as examples of pulmonary glanders were, in fact, but secondary processes consecutive to glanderous chancres of the nasal mucous membrane, and produced by emboli. But the bacilli may also be drawn from the lungs into the nasal cavities by the expired air, and the possibility of primary pulmonary glanders must not be rejected.

The skin constitutes a second mode of entrance for the pathogenic element. Cutaneous glanders, considered formerly as a peculiar form of the disease, is called *farcy*. Primary glanderous disease of the skin is only produced through wounds; farcy is thus

¹ It still remains doubtful whether air may so easily become the vehicle of the virulent agent. Eug. Renault, long before Cadeac and Mallet, never succeeded in transmitting the disease by making a healthy horse for several hours breathe the expired air of another affected by acute glanders, using for this purpose a linen tube which enveloped the nose of each animal. When we consider that it is always the immediate neighbor or the working-mate which is contaminated, we are rather led to think that, as a rule, there must nearly always be direct inoculation: that is to say, a deposit of virulent pus upon the respiratory mucous membrane or the skin, mostly excoriated. When experiments like Renault's have given but negative results it is not advisable to draw clinical observations with contrary conclusions.—L. T.

generally secondary, caused by emboli at the time of general infection. (Quite recently Babes, relying upon experimental results, has admitted the possibility of the penetration of the virus of glanders through the intact skin. In rabbits this author has made a friction upon the cutaneous integument with vaseline containing bacilli of glanders; in one case he obtained a specific ulcer followed by general infection. The bacilli penetrated into the pilous follicles, which they dilate by pullulation; after having passed the epithelial layers they pass into the lymphatic stomata.)

It is very rare for the bacillus of glanders to traverse the digestive mucons membrane. Viborg, Gerlach, Liautard, etc., have made horses ingest glanderous matter without obtaining any positive results; but of nine horses subjected to the same test by Renault, six became affected by glanders. In the few cases of glanders found in sucklings it is possible that the infection was not determined by milk, but by the inspired air. The dog, cat, and felines (lion, tiger, panther, bear) are mostly infected by eating glanderous meat; it is probable that wounds of the buccal mucous membrane are the starting-point of the bacillary invasion. In man, infection by eating meat from glandered animals has not been observed, notwithstanding that in numerous instances this meat had been consumed. The cases of infection consecutive to copulation are obscure as far as the manner of contamination is concerned. This may occur through the genital mucons membrane or by the respiratory mucons membrane (air). A few isolated cases of glanderous chancre have been observed upon the vaginal mucous membrane of the mare. As in tuberculosis, the bacilli may pass from the mother to the foetus.

The generalization of the glanderous process takes place in the same manner as that of the tubercular process. In the beginning it takes place regularly through the lymphatics. In the chronic form glanders sometimes remains stationary for a long time in the ganglions which are last attacked. By the circulation the bacilli are carried into all the organs. Finally, the glanderous centres in the bronchi may determine a secondary infection of the upper respiratory ducts, when the expired air carries virulent elements.

In acute glanders the bacilli enter the blood and the disease always becomes generalized.

Cadeac and Mallet claim to have demonstrated by experiments that the expired air of glanderous subjects is not virulent. According to these authors, the intra-tracheal injection of glanderous virus

does not produce any typic glanders, but only localized pulmonary and tracheal glanders. These facts do not appear sufficiently demonstrative.

Pathological anatomy of chronic glanders. Considered from an anatomical standpoint, this form of glanders is characterized by alterations which are due to chronic inflammatory processes—formation of granulations, suppuration, ulceration, cicatrization—which generally evolve upon the respiratory mucous membrane in the lungs, the lymphatic ganglions, the skin, and the subcutaneous connective tissue. The other organs are very rarely affected.

1. In the respiratory mucous membrane, which is most frequently the seat of glanderous lesions, they may assume two forms. We distinguish tubercular glanders with production of ulcers and cicatrices and *diffuse* or *infiltrated* glanders.

Tubercular glanders, which is the most common, is usually localized in the pituitary, in the upper regions of this mucous membrane, in the nasal walls, or in the turbinated bones. In the beginning we observe “pimples” which are of the size of a grain of sand or a millet-seed, of weak consistency, of rounded or oval shape, of whitish-gray or reddish-gray color; these little nodules, which are prominent upon the surface of the mucous membrane, are surrounded by a red zone. They are isolated or agminated; in this latter case they may reach the volume of a pea. Histologically, they are formed by an accumulation of lymphoid cells between which we find bacilli; these cells are soon affected by fatty degeneration; suppuration becomes established in the centre of the nodules, which become yellowish and ulcerated. The wounds thus produced are sometimes superficial “lenticular,” at other times deep, crateriform, with borders sharply cut; when several occur close together, which is not rarely, their borders are irregularly dentated; their base is lardaceous; sometimes they are found covered with a brownish crust. They may extend in surface and in depth, reach and invade the cartilages or the subjacent bones. Perforation of the nasal wall is thus produced, also maxillary tumefaction, exostosis of the turbinated bones, etc.

It is possible that the superficial lenticular ulcerations heal without producing any visible scar; but deep chancres always leave irregular, linear, or star-spangled, smooth or callous cicatrices, the form of which recalls that of the lesions which preceded them. Often the nasal mucous membrane and the turbinated bones are almost

entirely covered by these stigmata. Chancres and cicatricial islands also appear in the sinuses, the guttural pouches, and Eustachian tubes, where they may determine a lardaceous thickening of the mucous membrane and the accumulation of muco-purulent matter in its cavities; they are also found in the larynx, in the neighborhood of the aryteno-epiglottic folds (the process may even become extended to the arytenoid cartilages and lead to partial necrosis), in the trachea (the anterior wall of this organ often shows cicatrices and chancres which are of variable form and extent), and finally in the bronchi. Besides the ulcerations there always exists a catarrhal inflammation of the mucous membrane.

Diffuse or infiltrated glanders (glanderous infiltration) is marked by a catarrh of the nasal mucous membrane and its annexes, and accompanied by thickening of this membrane, superficial ulcerations, and peculiar star-like cicatrices, also venous thrombi and inflammatory infiltration of the submucous connective tissue.

2. In the lungs we also find both forms of the glanderous process. It is exceptional not to find any alteration in them. Of 52 glandered horses Bollinger found at the autopsy 4 with unaffected lungs. In the Berlin school they have been found unaltered in 10 cases out of 216, and at the Vienna school in 28 cases out of 173.

The *tubercular* form of pulmonary glanders is characterized by the existence of a more or less large number of nodules which vary in size from a millet-seed to that of a pea, and are of a gray, translucent or pearly color, surrounded by a hemorrhagic zone and marked in their centre by a yellowish-white point. Their diversity of aspect especially depends upon the age of the lesion: we find simultaneously young, caseous, calcified, and encysted nodules. The envelopment by a connective-tissue membrane is the result of reactional inflammation developed around the primary lesion. The mechanism of production of these nodules is variable. Sometimes they are of embolic origin; then we see them especially in the periphery of the lungs; their color is red in the beginning (infarctus); the largest have a conical form with peripheric base; their structure is the same as that of the lesions of the nasal mucous membrane. In other cases they represent small centres of lobular pneumonia; the alveoli are filled with red blood-corpuscles, leucocytes, and desquamated epithelial cells. Their central portion is soon softened, while in their periphery a secondary phlegmasia is developed, which, in the beginning, is marked by a whitish-gray inflammatory zone,

lardaceous and shiny, and which is transformed later into a connective-tissue capsule; these lesions may suppurate (cavities) or undergo calcareous infiltration. Finally, glanderous granulations often appear in the neighborhood of the primary centres in the tract of the inflamed lymphatics.

We also find alterations of chronic glanderous bronchitis: peribronchitis, para-bronchitis, bronchiectasis, atelectasis, gelatinous infiltration of the pulmonary tissue, and more rarely of circumscribed or exudative adhesive pleurisy.

Infiltrated pulmonary glanders has as an essential character tumors varying from the size of a nut to that of a child's head, and produced by a diffuse specific alteration of the alveoli and of the interstitial connective tissue (lobar centres or big bronchopneumonic lobular centres. On section these lesions often present the aspect of an encephaloid sarcoma; irregular in their disposition, they have a whitish-gray color, and are of a moist, gelatinous consistency. They may become indurated, form very dense connective neoformations (fibroid glanderous tumors of Gerlach) or break down into cavities.¹

In these two forms of pulmonary glanders the bronchial and mediastinal ganglions are hypertrophied, indurated, and invaded with specific granulations.

3. Cutaneous glanders or farcy is characterized by nodules, which are developed in the papillæ, derma, subcutaneous and intermuscular connective tissue. The farcy pimples, which vary in size from a juniper-berry to a pea, suppurate rapidly and become transformed into small chancreous wounds. The nodules of the subcutaneous connective tissue vary from the size of a pea to that of a hen's egg; there are inflammatory tumors (metastatic), which are soon followed by abscesses, the contents of which find an exit to the outside. Sinuous fistulous wounds are also produced with ulcerous characters, leading to a central cavity, and which allow the passage of viscid lympho-purulent discharge.

In the neighborhood of the tubercles the lymphatic vessels are inflamed; they frequently form hard knotty cords showing here

¹ It appears difficult to admit two pulmonary forms. There are always tubercles, and with this specific alteration, sometimes also induration of the interlobular and subpleural connective tissue, bronchial dilatations, etc., to a more or less marked degree; but all the lesions other than the tubercle—and there is no pulmonary glanders without it—are but accessory facts.—L. T.

and there tuberculiform swellings from the size of a lintel to that of a hazel-nut, which become ulcerated and transformed into chancreous wounds. The lymphatic ganglions are at first invaded by a parenchymatous phlegmasia; later they become indurated (connective-tissue hyperplasia); in their substance we find specific, whitish-gray granulations of the size of a pin's head, or yellowish centres which undergo caseous degeneration. In the beginning their shell is cellulo-fibrinous and infiltrated with small cells; later it is thickened (periadenitis and paradenitis).

In some rare cases chronic farcy leads to diffuse connective-tissue proliferation and an irregular, knotty thickening of the integument of the extremities, and especially of the head (glanderous elephantiasis or pachydermia).

4. Among the other organs the spleen is most frequently altered. It contains embolic nodules of the size of a millet-seed or pea—sometimes a little larger; they generally undergo calcareous infiltration; suppuration is very rare.

We may find similar neoformations in the liver, kidneys, testicles, brain, muscles, heart, and bones. In the bony tissue we observe a cellular infiltration of the marrow, tuberculiform granulations, and purulent destruction of the bone. Glanderous ulcerations of the gastric mucous membrane and vagina are very rare.¹

A microscopical examination of the blood shows a light leucocytosis (augmentation of the number of white corpuscles). In cases of acute general infection we find that it contains bacilli.

Pathological anatomy of acute glanders. Ulcerative destruction of the respiratory mucous membrane, a sero-bloody infiltration of the submucous, subcutaneous, and intermuscular connective tissue, inflammation or suppuration of the lymphatic vessels and ganglions, of the metastases in various organs, especially in the skin and the lungs: such are the essential anatomical alterations of acute glanders.

Upon the nasal mucous membrane we observe phagedenic chancres and *diphtheritic* lesions; the ulcerations spread rapidly, the pituitary is greatly infiltrated. The mucous membrane of the

¹ In somewhat old cases of glanders the red muscles are paler and the bones less solid. The ribs may easily be broken. In examining them, at first sight we recognize that their compact substance has lessened in thickness, and that the areolæ of the spongy tissue are much larger. All the bones have undergone a similar modification, which is more or less visible.—L. T.

larynx is the seat of a phlegmonous tumefaction which is particularly marked on the surface of the epiglottis and ventricles, where it is covered with chancres (laryngeal contraction); that of the pharynx shows similar lesions. The lungs contain metastatic purulent centres or recent tubercles. The skin is inflamed, strewn with pimples and chancres; sometimes we observê it in large splacelated blotches.

Symptoms of chronic glanders. The beginning of chronic glanders is often insidious and passes unobserved. It is to the absence of distinct manifestations in the initial stage of the disease that we must ascribe the inexact notions which formerly prevailed concerning the duration of the period of incubation (*latent glanders*). Experimental inoculation has shown that this incubation is from three to five days.

The first symptom is generally a unilateral nasal *discharge*, bilateral, mucous, whitish-gray (chronic nasal catarrh), the quantity of which is variable, and may even disappear at certain moments. Soon this discharge, which is formed of a viscous yellowish-gray or yellowish-green matter of bad aspect, coming from chancres, a matter which is mixed with a limpid, yellowish, catarrhal liquid. Quite frequently it is rendered bloody by slight epistaxis. These, which sometimes constitute the sole indicating symptoms of glanders, are due to ulcerative destruction of the small vessels and to a rupture of abnormally distended capillaries. In some cases the hemorrhage is considerable (it may be fatal when a large vessel of the lungs is ruptured inside of a cavity).

Upon the mucous membrane we generally observe *nodules* and *chancres*. These lesions, however, may appear a long time after the beginning of the disease. On the lower parts of the nasal wall and turbinated bones the glanderous "pimples" are sometimes perceptible to touch; their existence is of very short duration; they are soon transformed into chancres more or less deep, with den-tated, thickened edges, and with a lardaceous bottom which may become cicatrized, leaving after them fibrous islands of variable form.

The *tumefaction of the lymphatic ganglions* of the intermaxillary space is another important symptom. In the beginning the *gland* is diffuse, slightly doughy, and little sensitive; it is bosselated on its surface and adherent to the base of the tongue or to the lower maxillary; in some subjects it is adherent to the skin. In exceptional

cases the ganglionic tumefaction is wanting; in others, not less rare, the gland suppurates (superficial abscess formation).¹

General nutrition suffers in proportion as the affection progresses; it becomes more and more imperfect; emaciation appears, the hair is dull and bristly. We frequently observe the symptoms of asthma (pulmonary glanders), and the patients become rapidly fatigued. Frequently there is irregular febrile reaction, which is either remittent or intermittent, and the intensity of which is proportional to the number of ulcerations and the extent of the concomitant catarrhal affection. Sometimes violent efforts are followed by epistaxis. Glanderous lesions of the kidneys may be accompanied by hematuria (Fischer). When the disease is of long standing cedematous tumefactions appear upon the members, chest, and abdomen.

Farcy is more rare in the chronic than in the acute form of the infection; its favorite regions are the extremities, shoulders, chest, and abdomen. The pimples and tumors which are developed in the skin in the subcutaneous connective tissue vary from the size of a pea to that of a nut. In some instances these lesions disappear in part (flying farcy); at other times and most commonly they become transformed into crateriform sinuous chancres, which secrete a yellowish viscous liquid of bad aspect (farcy oil), sometimes blood. In certain cases we find small superficial ulcerations covered with scabs. The lymphatic vessels which originate from it are inflamed and tumefied (lymphangitis); later, they become indurated or form abscesses; these ganglionic alterations often induce lameness which persists for some time. In glanderous elephantiasis, generally quite rare, the skin is overrun with tubercles, wrinkled and very hard; sometimes the head has the aspect of the rhinoceros.²

¹ Ganglionic tumefaction may only be wanting when there is absolute absence of lesions in the nasal cavities, which is extremely rare. As to superficial abscess formation, it is still more rare in chronic glanders, and seems to constitute, when it takes place, a lesion which is foreign to the disease.—L. T.

² Under the name of *African farcy*, *Neapolitan farcy*, *river farcy*, *benign farcy*, *epizootic lymphangitis*, *farcinous lymphangitis*, *African lymphangitis*, authors have described a specific, parasitic, contagious lymphangitis which is determined by the *cryptococcus farciminosus* of Rivolta.

It is always consecutive to a traumatism, and takes place after an incubation of about three months; it is marked by multiple abscesses which are developed in the skin and the subcutaneous connective tissue, in the neighborhood of the inoculating wound; these abscesses are soon accompanied by a superficial or deep lymphangitis which ends in suppuration. Tuberculiform neoformations appear upon the skin, in the subcutaneous connective tissue, upon the tract of the lymphatic vessels, and in the corresponding ganglions, on the pituitary, the conjunctiva, in the muscular tissue,

Chronic glanders has a very slow *course*. The first distinct symptoms often appear only at the end of a few weeks, or even several months. Pulmonary glanders, after having existed for years without producing any appreciable phenomenon (latent, hidden, occult glanders), may be marked abruptly by multiple lesions of the skin or of the nasal mucous membrane, lesions consecutive to specific emboli. In the lungs the tubercles often develop a few days after the infection. Rest and nutritious food usually ameliorate the course of the disease and produce a marked improvement. Excessive work, cold, etc., lead to an aggravation of the trouble. The duration of chronic glanders is very lengthy; it may reach seven years.¹

and in the bones. They slowly undergo purulent destruction; those which are developed in the skin and the mucous membranes are transformed into fungous grayish wounds, which give exit to an oily or ichorous pus. These wounds do not present the ulcerous character of the glanderous lesions. The ganglionic obstructions lead to suppuration. Tubercles are never found in the lungs. As accompanying lesions we may find those of anemia, pneumonia, pleuro-pneumonia, pleurisy, and purulent infection.

When this affection is combated energetically it usually terminates in a cure within from one to seven months. In very serious cases it is generalized, and determines visceral lesions, when the patients die from these complications or exhaustion. Its mortality is about 11 per cent.

Epizootic lymphangitis and glanders may evolve simultaneously in the same animal (Peupion and Boinet).

The diagnosis is difficult. It may be established, however, by an attentive study of the symptoms and anatomical alterations. We may resort to inoculation; but the safest and most rapid way is to make a microscopical examination. The specific cryptococci exist in abundance in the pus and superficial layers of the wounds (Rivolta, Peupion, and Boinet). In an enlargement of 400 to 500 diameters the parasite appears as a large ovoid micrococcus with very refrigerent borders (Nocard).

The prophylaxis consists in preventing the infection of the wounds (aseptic or antiseptic dressing). The destruction of the altered tissue is the only efficient curative treatment. We may resort to cauterization, curettage, or extirpation.

Farcy of the ox, which is very rare in France at the present day, but quite common in Guadeloupe, has as the principal symptom a specific suppurative inflammation of the bloodvessels and lymphatic ganglions. The process may invade the viscera (spleen, liver, lungs), and determine serious emaciation, and lead to death. Farcy of the ox in Guadeloupe has no etiological relation with either glanders or tuberculosis. In specimens sent by Couzin to Nocard the latter has found a long, fine, entangled bacillus, which he has cultivated and inoculated in various species. Guinea-pigs inoculated with the cultures died within from twelve days to six weeks, according to whether the virulent matter is injected into the peritoneal cavity or under the skin. In the cow and sheep the evolution of the disease is much slower. The horse, donkey, dog, cat, and rabbit are refractory; in these animals only a small abscess is formed at the point of inoculation (Couzin: *Revue vét.*, 1879; Nocard: *Bull. Soc. cent. vét.*, 1888).—N. D. T.

¹ Besides these local phenomena there are important general symptoms: the cachectic state, adherence of the skin, dryness of the hair, and especially a very re-

Symptoms of acute glanders. Acute glanders is rare in the horse (10 per cent.); it is, on the contrary, the ordinary form in the donkey, mule, and hinny. Sometimes it appears from the onset, in other instances it follows the chronic form when this becomes generalized by other acute affections. It presents the characters of a septic infectious disease with rapid evolution, producing an ulcerous destruction of the respiratory mucous membrane and metastases in the skin, lungs, and other organs.

The disease begins with chills, intense fever, and hyperthermia, which reaches 42° C. We observe a muco-purulent, ichorous, bloody nasal discharge, often mixed with saliva and alimentary matters (regurgitation). The nasal mucous membrane is overrun with nodules and chancres which rapidly become confluent; it undergoes a true purulent destruction and becomes covered by a diphtheric exudate. These alterations of the pituitary may be developed in a very short time (two to three days). The respiration is laborious, rattling, wheezing, and moaning (laryngeal contraction). To these symptoms are often added those of farcy: cedematous tumefaction, tubercles, and chancres, lymphangitis (especially in the neighborhood of the head), inflammation and later abscess formation of the subglossal and pharyngeal lymphatic ganglions. Glanderous pharyngitis produces a marked dysphagia when it does not render deglutition altogether impossible; often, besides, anorexia is complete. We find also an abundant diarrhea; the urine contains a great quantity of albumin. The patients are extremely weak; emaciation progresses rapidly.

In general acute glanders has a sharp course; its usual termination is death. This ordinarily takes place within three to fourteen days. Glanders produced experimentally usually assumes the acute type.

Glanders of *cats* and *wild animals* (lion, tiger, etc.), produced by eating glanderous meat, almost always has a rapid course. Its principal symptoms are: acute inflammation of the mucous membrane of the nasal cavities, sinus, larynx, and trachea, a fetid discharge of greenish or bloody color, and a very pronounced dyspnoea, due to the obstruction of the submaxillary ganglions, tumefaction of the nose or entire head and legs (lameness), the appearance of

markable friability of the bones, which is notably marked by numerous fractures of the ribs.—L. T.

pimples and chancres upon the skin, sphacela of more or less extensive regions, diarrhea, emaciation, and exhaustion. Death occurred in from eight to fifteen days after infection.

The symptoms of *glanders* in *man* should be familiar to the veterinarian. While the receptivity of man for glanders is generally but little marked, a few cases of contagion are mentioned every year, mostly observed in veterinarians. The infection usually takes place by the hands, pituitary, lips, or conjunctiva. After a period of incubation of from three to five days the inoculating point becomes tumefied and painful, the lymphatic vessels and corresponding ganglions become inflamed. Sometimes the disease is marked by general symptoms. In nearly all cases we observe a nasal discharge and chancres upon the pituitary, pustules, and cutaneous abscesses, ulcerations of the buccal, pharyngeal, laryngeal, and ocular mucous membranes, articular tumefactions, intense fever, very marked general troubles, and sometimes a serious gastro-intestinal disease. In most subjects death occurs within two to four weeks; in other cases, more rare, the patients die within a few hours. Glanders may pass into a chronic state and be prolonged for months or even years. In acute glanders the blood contains bacilli (Wasseliëff).

The diagnosis is based upon the symptoms, also upon the probability of a contamination, upon the inoculability in the horse or guinea-pig, and lastly upon the detection of the bacilli.

While the affection is yet exclusively local we may obtain a cure by deep cauterization. We have recently observed such a result. As soon as the glanderous infection is generalized all treatment is futile.

Clinical diagnosis. In the horse there is no disease of which an exact diagnosis is so important as that of glanders, and in no other does the differential diagnosis present such great difficulties. In order to recognize glanderous infection we may employ several processes :

1. A clinical examination.
2. The use of the rhinoscope.
3. Trephination of the sinus.
4. Amputation of the ganglions of the submaxillary region.
5. Administration of pyretogenic agents.
6. Auto-inoculation.
7. Inoculation to other species.
8. Culture of the bacillus.

1. The *clinical characters* enable us to affirm the existence of

glanders when they are found in sufficient numbers : unilateral, limpid, viscous, or purulent nasal discharge ; nodules and chancres with thickened borders and lardaceous bottom are developed upon the nasal mucous membrane ; a hard gland, bosselated and fixed to the base of the tongue or appearing adherent to the bone ; pimples, chancres, and farcinous tumors upon the skin ; knotty lymphangitis ; phlegmonous tumefactions occurring without any apparent cause upon the legs, belly, chest, testicular pouches, etc.) ; the hair is dull, bristly ; emaciation, febrile asthmatic spells, cough, periodical epistaxis, asthma, depression of strength. But often most of these symptoms are wanting ; in some patients but one is observed. In these cases we say that the horse is under *suspicion* of glanders. Among the symptoms which confirm suspicion we must particularly mention unilateral and bilateral nasal discharge which presents no special characters, and a hard, embossed, elongated, painless gland. A ganglionic hypertrophy, which is quite similar, may be observed in chronic strangles. Formerly it was thought that the glanderous gland did not form abscess ; this assertion is erroneous, for the submaxillary ganglions sometimes undergo purulent disintegration. Chancres, which are well characterized, even when existing alone, have a high diagnostic value (we may observe chancres without any nasal discharge). It is important not to confound them with accidental wounds of the mucous membrane, which ordinarily present a linear form and are covered with a scab. It is always advisable to examine the internal fascia of the wing of the nose ; it is frequently the seat of ulcerations. The nodules and ray-shaped cicatrices, which may also be scattered like stars, are also important lesions from a diagnostic standpoint. The marks left by traumatism of the pituitary are elongated or irregular.

A continuous febrile remittent or intermittent reaction, occurring without any visible cause, should cause the horse in which it occurs to be suspected. The same is the case in emaciation, cough, asthma, a dull and broken state of the hair, when circumstances point to a probability of contamination. If observed in animals belonging to crowded stables, these symptoms require attentive watching.

2. An examination of the nasal cavities by means of the *rhinoscope* has been practised for a long time in veterinary medicine. The upper regions of the pituitary are not visible to ordinary inspection. This instrument may render great service in the diagnosis of glanders.

3. *Trephination of the maxillary sinus* was first recommended by Hertwig in 1841, then by Haubner in 1859. It permits us to recognize glanderous lesions developed in the sinus. This process of exploration, however, has not the value ascribed to it by authors: quite recently the mucous membrane of the sinus remained unaffected during the course of glanders; on the other hand, apart from this affection, we may find it inflamed and thickened to a considerable extent. The glanderous lesions which, according to Haubner, are likely to appear upon the operated wound, do not always occur, even when specific alterations exist in its neighborhood.

4. *Extirpation of the submaxillary ganglions* has been practised with the object of making an anatomical examination of these organs (Haubner, Bollinger). In many cases this method furnishes only doubtful information: the characteristic glanderous centres are not constantly seen; the ganglions may become indurated in simple chronic nasal catarrh; we frequently find purulent islands in them in cases of strangles; and we may finally recognize sarcomatous and carcinomatous metastatic deposits. On the contrary, when the *gland* contains glanderous granulations the bacillus may easily be discovered in it. A bacteriological examination (staining and culture of the pathogenic agent) constitutes then a valuable *intra-vitam* diagnostic method (Rieck).

5. *Artificial production of fever* was advised by H. Bouley in 1843, in order to produce the acute type in latent glanders. Experience has demonstrated that chronic glanders frequently takes an acute course when the organism is under the spell of another febrile disease. More recently Lustig has advised submitting the suspected animals to fatiguing work, which may determine oscillations of the temperature and nasal hemorrhages. According to Cagny and a few other veterinarians, subcutaneous injections of essence of turpentine made in horses affected with chronic glanders provoke acute manifestations.¹

6. *Auto-inoculation (malleosation)* was formerly performed by inoculating suspected animals upon a healthy part of the pituitary with their own discharge or with the product of their ulcerations.

¹ The results obtained with tuberculin have induced researches with the object of enlightening the diagnosis of pulmonary glanders by inoculation of the products derived from the glanderous bacillus. According to Helman, of St. Petersburg, *malleine* injected under the skin in a dose of 1 cubic centimetre produces distinct symptoms pointing to this affection in cases of latent glanders.—N. D. T.

It has given very divergent results. In numerous experiments made by Bagge, Tscherning, Saint-Cyr, and others, at times a glanderous process was developed upon the inoculating wound, at other times the virulent insertion remained sterile. It seems that glanders creates a certain immunity from auto-inoculation.

7. *Inoculation in other animals* is evidently the most valuable method for a safe diagnosis. The donkey, horse, and guinea-pig are the best agents; then come the dog and the rabbit; the latter is of little use. In order to obtain the pure bacillus Löffler recommends the inoculation of the prairie dog and field mouse; Kitt uses in preference the burrow rat and the hedgehog. Of all animals the donkey is the most susceptible to the action of the glanderous virus; in this animal glanders has an acute evolution and terminates in death within eight to ten days; unfortunately this animal is difficult to obtain. After the ass comes the horse, which ought to be used, if it is possible to obtain one of little value. Let us add that in this animal negative results do not permit us to conclude positively the non-existence of glanders; some old horses affected by latent glanders are refractory to inoculation.

For the requirements of practice Löffler, Nocard, Cadeac, and Mallet have advised the guinea-pig. In this animal inoculation invariably succeeds and the disease is marked by symptoms which are quite characteristic. In the male the glanderous orchitis is typical.¹ The duration of the disease is about one month. According to Löffler, inoculation must be performed in the following manner: the hair should be cut upon the lateral surface of the abdomen; make a cutaneous incision about one centimetre in length; then, by means of a sterilized needle, lacerate the subcutaneous connective tissue and inject the suspected matter into the pocket thus made. The first symptom is redness of the edges of the wound, which is soon transformed into a round or ovoid ulcer with thick borders, of bad aspect. Toward the end of the first week the

¹ Injection into the peritoneal cavity of a male guinea-pig of suspicious products permits us to make a rapid diagnosis of glanders. If we have to deal with glanderous matter, as soon as the second or third day we observe a tumefaction of the testicles and an abnormal tension of the membrane of the scrotum; at this time the vaginal covering is already the seat of a suppurating phlegmasia, and the pus contains the specific bacillus. Orchitis and vaginitis are precocious and typical lesions of glanders. (Strauss: Roque de Silveira, *Compt. rend. de la Soc. de Biologie*, 1889-91). When the injected matter comes from a nasal discharge or any other impure product the guinea-pig may die in twenty-four hours from peritonitis. Thus, it is prudent to inoculate at the same time several subjects.—N. D. T.

lymphatic ganglions become tumefied and suppurating. Sometimes the whole process retrocedes from that date. Orchitis ordinarily appears in the course of the second week; we recognize hard nodes in the testicles, which form abscesses and discharge pus which swarms with bacilli. We observe, besides, purulent arthritis. In the skin and subcutaneous connective tissue pimples appear which also form abscesses (upon the fascia these glanderous lesions often start from the periosteum; they perforate the bones and open in the nasal cavities). Respiration is hindered; the nostrils give exit to a discharge which dries up around these orifices. Generally the guinea-pig, which is much emaciated and exhausted, dies in the course of the third or fourth week; in some cases death occurs within eight days; in others it takes place toward the end of the second month only. At the autopsy we find nodules in the lungs, spleen, epiploon, testicles, and under the peritoneum, also chancres upon the pituitary, a perforation of the nasal wall, and partial destruction of the bones of the face. The blood does not contain any bacilli, but they are found in the altered tissues. (According to Cadeac and Mallet, inoculated glanders may take a chronic course and produce caseous tubercles in the lungs, the heart, liver, spleen, lymphatic ganglions, etc.; its duration is then two to four months; sometimes it ends in a cure.)

The DOG has been recommended as an agent in doubtful cases (Pütz, Galtier, Violet, Reul, Molkentin, and others). Inoculation is made by means of a seton impregnated with suspected secretions and passed under the skin of the neck.¹ On the third day there are developed upon the orifices of the tract ulcerous wounds with purulent base and indurated borders, which become gradually extended, taking an irregular form and accompanied by distinct general symptoms. In a majority of cases these are the only phenomena observed. In some instances we notice tubercles and chancres, in other regions of the integument acute arthritis (especially upon the hind legs), also a bloody diarrhea, purulent conjunctivitis, ulcerous keratitis, emaciation, etc. Out of 300 virulent inoculations made by Neimann only four remained sterile.² However, on account of the rarity of general symptoms and the sometimes equivocal

¹ There is another more simple procedure which consists in making superficial scarifications on the skin and the frontal region and depositing the suspected products in them.—N. D. T.

² Neimann (communicated note).

characters of the local ulcerations, the dog should only be used when animals more sensitive to the announcing inoculation are wanting.

Formerly the *rabbit* was often used, but after inoculation he dies more frequently from septicemia than from glanders. In many cases the animals must be observed for two months (Friedberger), and the results do not always allow us to form an opinion. When we pass threads soaked in suspected matter through the internal integument of the auricular shells we soon observe local abscesses, varying in size from a hempseed to a pea; later, irregular wounds, with rugous, purulent base and thick borders; they perforate the concha at times; in other instances they heal. Friedberger considers these lesions as little characteristic. Later we observe a nasal discharge if the inoculation has been successful. Death occurs within two months. At the autopsy we find the lungs and spleen studded with glanderous nodules.

In order to isolate the bacillus Löffler has advised the use of the *prairie dog*. But when the inoculations are made with nasal secretion all the animals die from septicemia. When inoculated with cultures they die within three or four days. On the first day (sometimes on the second) the animals are apparently healthy, then they become depressed, stop eating, the hair is bristly, respiration becomes embarrassed, and death occurs suddenly, without convulsions. At the autopsy we find at the point of inoculation a greenish or whitish-gray infiltration. The lymphangitic cords start from this point and lead to the corresponding ganglions, which are tumefied and marked by numerous yellowish-white granulations. The liver contains grayish nodules. All these lesions contain numerous bacilli. Ordinarily the lungs are unimpaired. The nasal mucous membrane and the skin are in a normal condition. The inoculation is made, as in the guinea-pig, by introducing suspicious matter into a little subcutaneous pouch which is hollowed in the dorsal region.

According to Kitt, we may also inoculate with cultures the *burrow rat* (*Arvicola terrestris*), an animal in which the inguinal ganglions and the spleen are the organs of predilection of the glanderous bacillus. This author has also advised the use of the hedgehog (*Erinaceus europeus*); in this subject inoculation determines, in the spleen and lungs, characteristic lesions. Finally, Krausfeld and Grünwald have recognized that glanders is transmissible to the spermophile (*Spermophilus guttatus*).

8. *Bacteriological search for the bacillus* is the last resort to insure a diagnosis. Unfortunately, as shown by Löffler's experiments, it cannot be made in all cases. We obtain only characteristic cultures with the contents of closed glanderous centres (farcy pimples, pustules of the pituitary in the entrance of the nasal cavities, lymphatic ganglions); cultures upon serum appear as transparent yellow drops; on potato they form a coating of a honey-yellow color. In practice it is quite difficult to collect the culture-matter pure; often we succeed only by removing a *gland*. Examination of the product of secretion of chancres or of the nasal discharge is not of great importance; the bacilli, in fact, have no special histo-chemic reaction, and these products contain an abundance of other bacteria, which behave in the same manner as the glanderous bacillus toward staining agents. The secretions are frequently decomposed; the culture of the microbe is not successful or is very difficult. As a last resort we may practise inoculation in the guinea-pig. Löffler advises the inoculation of 3 to 5 guinea-pigs—preferably males; he recommends also the use of suspected matter which has been obtained at various periods, the nasal discharge not being equally infectious at all times. It is not sufficient to perform inoculation by a simple puncture with the lancet; we must dig a subcutaneous pouch and deposit a considerable quantity of discharge in it. The negative results observed after repeated inoculations upon several guinea-pigs have, from a diagnostic point of view, the value of a positive result.

STAINING THE BACILLUS. 1. Löffler at first recommended the following method: the preparation upon lamella is placed for five to ten minutes in a solution of methylene-blue (30 c.c. of a concentrated alcoholic solution of methylene-blue are dissolved in 100 c.c. of a solution of potassium at 1 per 1000); then for a few seconds in a 1 per cent. solution of lactic acid; it is then washed with distilled water and mounted.

2. More recently he has advised the use of a solution of gentian-violet in Koch-Ehrlich's aniline solution, with the addition of an equal quantity of a solution of potassium at 1 per 10,000, or of an ammonia solution at 0.5 per cent. The preparation is left floating in it for five minutes; it is then held for one second in a 1 per cent. solution of acetic acid, to which is added a sufficient quantity of an aqueous solution of tropeolin 00, in order to give to it a yellow Rhine-wine color; it is washed with water immediately after and mounted.

Clinical differential diagnosis. Glanders may be confounded with numerous morbid states, among which we will particularly mention the following :

1. *Simple chronic nasal catarrh.* This is marked by a persistent nasal discharge, by tumefaction of the lymphatic ganglions of the submaxillary, and sometimes by superficial erosions of the pituitary. Formerly it was given the name of *suspicious glanders*, and it was generally considered as the first stage of glanders. An accurate diagnosis can only be made after a long period of observation. In nasal catarrh there are usually no cicatrices upon the mucous membrane, and a cure is obtained by local treatment. Inoculation in the guinea-pig removes all doubts.

2. *Chronic catarrh of the sinus and guttural pouches.* In order to differentiate glanders from these diseases it is sometimes necessary to watch the animals for a certain time. Trephination clears up the diagnosis and permits the drying up of the purulent secretion of the mucous membrane of the sinus. In case of dental caries we have as a guide examination of the teeth and the fetid odor which escapes from the buccal cavity. In one patient Siedamgrotzky has found at the same time dental caries, chronic catarrh, and glanders of the sinus. In some cases inoculation alone may confirm the diagnosis.

3. *Strangles.* During the course of strangles we observe frequently lymphangitis and cutaneous ulcerations upon the head, neck, and other regions. But the character of the pus, the course of the disease, and its rapid cure permit a differentiation. The chronic form which is accompanied by metastases exposes the error so much more.

4. *Follicular ulcerations of the nasal mucous membrane.* These heal rapidly without leaving any cicatrices ; we observe them frequently upon the skin in the neighborhood of the nostrils, and ganglionic tumefaction is generally little marked.

5. *Contagious pustular stomatitis.* This is usually localized upon the buccal mucous membrane, and is characterized by its mildness.

6. *Simple inflammatory lesions of the nasal mucous membrane,* or those which are of traumatic origin and determined by various influences (hot vapors, smoke, foreign bodies, erosions which have been made with the nails, etc.). They may be easily recognized. In general, the cicatrices which they leave are quite striking by their location toward the lower regions of the nasal cavity (wall),

by their thickness, and by their elongated linear or angular form ; they are sometimes found upon both fascia of the wall in corresponding points.

7. *Simple epistaxes* consecutive to traumatic actions, non-specific phlegmasias, aneurisms, angiomas of the mucous membrane, pulmonary hyperemia, and pneumonia. Attentive observation, sufficiently prolonged, permits us to make the distinction (see Hemoptysis).

8. *Leucemia*. This disease often presents a striking clinical analogy to glanders (emaciation, weakness, glandular tumefaction, epistaxis, intermittent fever, tumefaction of the extremities and abdomen, etc.). Often the diagnosis is only possible by a microscopical examination of the blood, which shows a considerable increase in the number of white corpuscles. In glanders the number of leucocytes is not greater than in the normal state. Leucemia producing no morbid secretion, inoculation could not be made.

9. *Tumors of the nasal cavities*. These are sometimes only recognized at the autopsy. The most common are angioma of the nasal wall, sarcoma, carcinoma, polypi, and benign connective-tissue proliferations. These latter may simulate infiltrated glanders. Invasion of the lymphatic ganglions of the submaxillary by cancerous elements increases the difficulties of the diagnosis.

10. *Phlegmonous tumefactions* of the extremities [penetrating disease (Enischuss), elephantiasis] of the head or other regions, and pyemic metastases which they produce. They may mislead the practitioner in some cases. The existence of a primary lesion (wound, contusion, etc.) and the mode of development of these affections put us on the right path.

11. *Urticaria*. This is sufficiently characterized by its mild cutaneous lesions, which never form abscess, are not accompanied by ganglionic tumefactions, and disappear rapidly.

12. *Petechial fever*. This is distinguished from acute glanders by the slight intensity of the fever and the extent of cutaneous tumefactions.

Anatomical differential diagnosis. On this question, belonging to pathological anatomy, we must be restricted to describing the principal pulmonary alterations which may be confounded with glanders. Let us observe from the onset that the macroscopic and microscopic recognized facts are not sufficient in all cases to give a final decision ; we must frequently take into consideration the etiological and clinical facts.

1. In old animals we frequently find in the lungs *caseous*, *calcified*, and encysted *nodes*, which are of the size of a lentil to that of a pea; these are sometimes the remains of pneumonia by foreign bodies; in other instances alterations which are consecutive to inhalation of dust. These pneumonic centres may exist in large numbers. They are distinguished from glanderous nodules by their uniformity of aspect, their equal age, and the absence of other lesions of glanders, and especially by no alteration of the bronchial ganglions. They may contain particles of vegetables or other foreign bodies. In one case Martin has found there myceliform filaments (pneumomycosis).

2. *Embolic lesions* of the lungs produced during the course of strangles, pyemia, etc., vary in size from a pea to that of a hen's egg; sometimes they are found scattered in the pulmonary parenchyma, and are especially abundant in its superficial layers; all have the same aspect and the same age; in the beginning they are simple hemorrhagic infarcts of conical form. But these peculiarities are also observed in the embolic glanderous nodules, and often the diagnosis must be based upon the absence of other alterations of glanders or upon the existence of a primary morbid centre independent of this affection.

3. *Pulmonary tuberculosis* is essentially characterized by the presence of Koch's bacillus. According to Csokor, the bacilli of glanders, which are larger than those of tuberculosis, seem constituted by a series of joints which are alternately clear and dark. Glanderous nodules are especially formed by lymphoid elements, while the tubercles represent true granular tumors in which the microscope shows three varieties of cells (giant, epithelial, and lymphoid cells). Finally the pulmonary tubercles are frequently accompanied by similar alterations of the serous membrane.

4. *Sarcomata* and *carcinomata* of the lungs may easily be recognized at the microscopical examination.

5. *Numerous peribronchitic centres* which, according to Dieckerhoff, are located in the walls of the bronchioles and form small nodosities from the size of a grain of sand to that of a lentil, are hard, whitish-gray; in the course of time they become calcified and encysted; we recognize their nature by their uniform dimensions, by the absence of other lesions which may be connected with glanders, and particularly by the normal condition of the bronchial ganglions.

We must also point out the bronchitic and parabronchitic process in general, bronchiectasis, multiple foetal atelectasis, and chronic interstitial pneumonia. In all these cases we observe neither the nodules of glanders nor the other lesions common to this disease.

In old horses we frequently find in the liver and spleen numerous small calcified, encysted islands of variable dimensions which exist independently of any glanderous lesion. They are produced by emboli coming from the intestine, and are mostly of vegetable origin.

Treatment. Spontaneous healing of glanders is possible, like that of tuberculosis, but it is extremely rare. The cases of healing by medicinal agents are generally based upon diagnostic errors. At various periods and especially since 1860 authors have tried a large number of therapeutic agents: chlorides, bromides, iodides, salts of copper, mercury, silver, iron, arsenic, strychnin, phenic acid, alcohol, etc. Levi is said to have obtained good results by intra-tracheal injections of Lugol's solution (iodine, 2 parts; iodide of potassium, 10 parts; water, 100 parts. Trinchera and other authors have demonstrated that these injections, instead of being advantageous, favor the glanderous process and precipitate its course. The treatment of cutaneous glanders by the actual cautery is also inefficient, because it is applied to secondary metastatic lesions. We must, therefore, be very careful concerning cures of cases of farcy.

Preventive inoculation, which has been recommended by Bagge and Tscherning, has only a historical interest.

Glanders can only be combated with any efficiency by prophylactic means and by a strict application of the sanitary police measures which prescribe laws and regulations in force in various countries.

It is considered as a disease setting aside a contract of sale in Prussia, Bavaria, Würtemberg, Baden, Hessen, after a delay of fourteen days; in Saxony and Austria, after a delay of fifteen days; in Switzerland after twenty days.

CONTAGIOUS PLEURO-PNEUMONIA.

HISTORY. The first writings relating to contagious pleuropneumonia date from the end of the seventeenth century. In 1693 it was observed in the province of Hessen. It became widely extended in the beginning of the eighteenth century, and had an

epizootic course at this period in Switzerland and the neighboring countries: Würtemberg, Baden, and Alsace. The publications we possess concerning this first epizootic date back to the year 1743. In 1751 the Sanitary College of Zürich attracted the attention of owners of live stock to this disease by publishing a pamphlet with a description of its principal symptoms. In 1773 the naturalist, Haller, gave a description of it, in which we find quite exact descriptions of its manifestations, its alterations, nature, and the sanitary police measures necessary. It made its appearance in England in 1735, and in France in 1765. From 1790 it spread in Germany, France, and Italy. Since the beginning of the nineteenth century it has invaded all Western Europe. About 1840 it was introduced into America, Africa (Cape), and Australia.

Etiology. Pleuro-pneumonia is an infectious disease of the ox, which is essentially characterized by specific inflammation of the lungs and pleura. It affects, though exceptionally, the subjects of other domestic species.

Its only cause is contagion. The doctrine of spontaneity, which formerly was generally accepted, has been entirely abandoned at the present day. Willems, Zürn, Hallier, Weiss, Pütz, Süssdorf, Brazzola, Bruylants and Verriest, and Lustig,¹ have begun researches

¹ In 1885 Lustig found in the lymph of newly-inflamed parts: 1, a bacillus which liquefies gelatin; 2, a micrococcus the colonies of which resemble the white of an egg when cooked; 3, another micrococcus which is different from the preceding in the golden yellow color of its cultures; 4, a third micrococcus the gelatin culture of which is similar to a dark-yellow wax seal. In 1886 Cornil and Babes tried in vain to discover the pathogenic agent of the disease. As early as 1884 Arloing begun researches which he has continued for five years, and the results of which were published by him in 1889. Gelatin cultures of the serum pleuro-pneumonia have given colonies from which he has been able to draw four different microbes: 1, a bacillus which rapidly liquefies gelatin (*Pneumo-bacillus liquefaciens bovis*); 2, a non-fluid producing micrococcus, the white colonies of which resemble drops from a white wax candle (*Pneumo-bacillus guttaceri*); 3, a micrococcus the white colonies of which are spread in a thin layer and show wrinkles and folds after a time (*Pneumo-bacillus lichenoides*); 4, another micrococcus the colonies of which are elongated or circular, and take a beautiful orange-yellow tint (*Pneumo bacillus flavescens*). The first two may be aerobic or anaërobic; both of the latter are exclusively aerobic. The inoculation experiments made with cultures of these micro-organisms appeared to establish that the *Pneumo-bacillus liquefaciens* is really the pathogenic agent of contagious pleuro-pneumonia. (See Arloing: Journ. de Lyon et Recueil vét., 1889.)—N. D. T.

[The *Pneumo-bacillus liquefaciens bovis* was first isolated and described by W. L. Zuill while a student in the Medical Department of the University of Pennsylvania in October, 1883. The description then given of this germ has been since corroborated by the more recent and exhaustive researches of Lustig, Cornil, Babes, Arloing, etc., who made inoculation experiments and produced pleuro-pneumonia thereby. American Vet. Review, vol. ix., 1885-86.—W. L. Z.]

with the object of determining the infectious agent. In 1886 Pöls and Nolen (of Amsterdam) thought they had discovered it. They constantly found in the pulmonary exudate micrococci of a diameter about 0.9μ , which were sometimes isolated, and in other instances gathered in small chains of three to six. In the unstained preparations the *cocci* appeared surrounded by a very distinct capsule, slightly colored, which was wanting in the cultivated microbes. The cultures upon plates at the temperature of the room showed, within twenty-two to twenty-five days, white colonies with whitish shiny reflex, slightly prominent and distinctly defined. The cultures obtained by puncture produced nail-shaped colonies, which soon took a creamy, then yellow color. Development of the *cocci* is arrested after exposure for one hour at a temperature of 67° C. If injected directly into the lungs, the cultures have produced in the ox and in the dog, guinea-pig, and rabbit an extensive pneumonia; the exudate contained the characteristic micrococci. Out of 100 animals inoculated with cultures and then exposed to contagion by being placed among pleuro-pneumonia subjects, not one contracted the disease.

The infection of healthy stables takes place through sick or infected animals which are brought into them, more rarely by the intermediation of man. All subjects are not equally susceptible to the contagium; we generally estimate the proportion of those which appear to possess immunity at 25 per cent. In animals of the same stable the disease is mainly propagated by the air; it is transmissible from its beginning, when there is as yet no marked symptom to be observed. The infectious virulence reaches its maximum at the height of the disease. Contagion may take place from eight to ten weeks, and later, after cessation of the epizootic or the apparent cure of the affected animals; this occurs when the necrotic centres exist in the lungs, exactly as in infectious pneumonia of the horse. Propagation through the air may take place from a great distance (50 metres and more). Contamination often occurs through several intermediaries [attendants, utensils, food, dog, etc. (Dubos)]. The meat of slaughtered animals is very rarely contagious; it seems to lose its virulence on becoming cold.

The infectious agent possesses great resistance to destructive agents. In some mediums it may be preserved for three or four months and even longer. The incubating period lasts, on an

average, from three to six weeks¹ (the minimum is eight days, the maximum three months). A first infection may confer immunity for the remainder of life.²

AFFECTED ANIMALS. After cattle plague, pleuro-pneumonia is the most fatal disease of the bovine species. When it appears in a stable it may have the course of a sporadic affection; in a majority of cases, however, it exists from the onset as an enzootic or epizootic disease. It is stationary in some parts of Holland, Belgium, England, and in a few provinces of Germany, in Africa and in Oceanica. The countries of Oriental Europe are almost free from it. In Russia it is observed only on the western frontier and in the seaports. It is especially common in crowded stables, where cattle are frequently changed, and in localities situated upon main roads; it is rare—almost unknown—in countries where the importation of animals of the bovine species is light.

The charts furnished by the sanitary service for the years 1886–87 demonstrate that pleuro-pneumonia existed in an intense form in central Germany, mainly in the lowlands, north and east of the Harz Mountains, between the rivers Saale, Elbe, and Leine. The districts of Magdeburg, Merseburg, Erfurt, Hanover, Hildesheim, Lüneburg, and the Duchies of Brunswick and Anhalt were its principal centres.

The losses occasioned by pleuro-pneumonia are enormous. In Great Britain nearly 200,000 animals died in 1860; for a period of six years the mortality reached nearly 1,000,000 (a loss of 50,000,000 dollars). In Oceanica, from 1860 to 1872, it killed 1,500,000 animals. In France, in the Department of Nord alone it destroyed more than 200,000 subjects in ten years; Holland lost 600,000 cattle from 1830 to 1840, and Rhinish Prussia 100,000 from 1835 to 1845. Lately its ravages have been much less on

¹ Virus of pleuro-pneumonia which is obtained pure from the lungs and preserved in tubes loses its virulent properties but slowly (Pasteur). It may be found directly in this organ by means of a sterilized tube-pipette, after having cauterized the pleura at the point where this tube is introduced, or use Laquerrière's dilating trocar. This virus possesses great resistance to the action of cold. Fragments of lungs affected by pleuro-pneumonia, or the entire lobes exposed for one year at a temperature of 5 to 6° C. in a refrigerator, have retained their virulence. Inoculations which were made with the serum obtained from them, and with that taken from a cow suffering from pleuro-pneumonia which had been killed just before, have given nearly the same results. (Laquerrière: *Bulletin de la Société Vétérinaire*, 1889–90.)—N. D. T.

² The duration of immunity, which varies according to the subjects, generally lasts for several years. It may continue during life.—N. D. T.

account of the preventive measures taken by the Governments. According to one of Fleisch's statistics, in Prussia, from 1876 to 1886, 23,582 oxen died of pleuro-pneumonia, or were killed because affected with this disease. The figures corresponding with each of the ten years are as follows: 1876, 3117; 1877, 1980; 1878, 2098; 1879, 2364; 1880, 1749; 1881, 1982; 1882, 2079; 1883, 3070; 1884, 3252; 1885, 1891. In all provinces, with the exception of Saxony, pleuro-pneumonia has diminished gradually. The amount of indemnities allowed during that period was 1,062,500 dollars.¹

The disease has also been observed in the goat (Spinola, Koppitz, and others), buffalo, bison, and yak. Cases of "pleuro-pneumonia of the pig" are based upon errors of observation. The same is true concerning instances of contamination of man, and especially of children. The existence in the latter of a pneumonia presenting anatomical alterations analogous to pleuro-pneumonia, although interesting, is without any value in determining the communicability of this disease to the human species. Man may eat the meat of affected animals without bad results.

Pathological anatomy. Pleuro-pneumonia is characterized anatomically by progressive interstitial pulmonary phlegmasia, with secondary hepatization of the lobules and extension of the morbid process to the pleura. In the majority of cases only one lobe (ordinarily the left) is affected. The lesions vary with the duration of the disease.

1. In the beginning the lungs present small circumscribed in-

¹ In France, during the year 1887, pleuro-pneumonia was observed in 31 departments: 9 had less than 10 cases; 12 from 10 to 25; 2 from 25 to 50; 5 from 50 to 100, and 3 more than 100 (Meurthe et Moselle, 118; Nord, 337; Seine, 455). The number of animals having died of pleuro-pneumonia, or which were killed because affected with this disease, was 910 for these three departments, and 693 for the rest of France. The disease forms three principal centres of origin which are more or less old: one in the extreme north, one comprising the department of Seine and the neighboring department, and one in the extreme southwest; there are two other secondary centres, one in both Savoy provinces, the other comprises a few departments of the west. The Department of Meurthe-et-Moselle, which had not had one case of pleuro-pneumonia since 1883, was infected toward the end of 1886 by animals coming from Paris. In almost all departments the disease has decreased since the promulgation of the sanitary police laws. Indemnities were allowed for 3571 animals in 1882, and for only 1454 in 1887; the total of these indemnities was 688,500 francs in 1882, and 323,908 francs in 1887. The mortality consecutive to inoculation also diminished in a marked proportion: in 1882 it was 192 for 13,669 animals (1.4 per cent.); in 1887, of 55 for 7911 (0.7 per cent.). (Tisserand: Rapport sur le Service des Epizooties en 1887, in *Recueil Vét.*, 1889.)—N. D. T.

flammatory centres, which may be of the size of a hazel-nut or walnut. The interlobular tissue is hyperemic, studded with hemorrhages and infiltrated with serum; the red hepatized lobules are surrounded by bands, which are from one to two millimetres wide, of clear shade, and filled with serous or lymphatic liquid. When these centres are located superficially the pleura on their surface is opaque and covered with small flakes.

2. At the height of the disease we find a lobar pneumonia and a secondary pleurisy: The lungs are distended, hard, very heavy (their weight reaches as much as fifty kilogrammes); they sink in water and do not crepitate on incision. Sections made in its thickness have a marbled aspect. The interstitial and thickened connective tissue forms yellowish-red or whitish-gray bands, which surround the lobules, the shade of which is dark, and having a thickness from two millimetres to two centimetres. The color of the islands which they circumscribe depends upon the age of the hepatization; it varies from brown-red to gray-yellow. Newly-affected lobules are blood-red, brown-red, or dark-brown (red hepatization); those already affected for some time are colored light-red or yellow (yellow hepatization); the oldest show a gray coloration (gray hepatization). The deep regions of the lobules are generally those marked by yellow or gray hepatization. We may find encysted lobules, the tissue of which is normal or compressed; others are only hyperemic.

If we examine carefully the interstitial track, they appear formed in the beginning by an œdematous infiltration, which later becomes gelatinous fibrino-plastic, and increases gradually in consistency, and may be transformed into connective tissue. The lymphatic spaces have undergone lacunal dilatation, and are at first filled with a serous or fibrinous liquid. In robust animals the alveoli contain a very dense croupous matter, with an apparently granulated section; in subjects having weak constitutions the exudate is serous. Formerly, when pleuro-pneumonia was characterized by croupous hepatization, it was characterized as *synchous*; when the exudate was serous the disease was designated as *torpid*.

The lymphatics of the lungs are dilated and thrombosed; their walls are thickened by cellular infiltration. The bloodvessels also show thrombi and small hemorrhagic infarets. The bronchioles contain a fibrinous matter with numerous leucoeytes. The bronchial and mediastinal ganglions are tumefied.

The pleura is covered with false fibrinous membranes or granular masses, which are soft and become easily detached. This exudation may reach a thickness of two centimetres; its surface frequently shows a reticulated aspect. The serous membrane is injected, ecchymosed, rugous, and irregular upon its surface. In the pleural pouch we find besides a variable quantity of clear or suspicious liquid, which is generally odorless, and holds in suspension flakes or clots of a certain size.

3. In more advanced periods the hepatized pulmonary regions may return to their primary state or become indurated, undergo caseous degeneration or calcareous infiltration; they may also be overrun by suppuration or affected by necrosis. In the latter case the mortified lobules produce a suppurating inflammation in their periphery; they are soon separated from the adjacent tissue; they constitute true sequestra, which are contained in cavities surrounded by a smooth fibrinous membrane. Thus isolated, the necrosed portions may preserve their primary characters for a long time; they also become frequently softened and transformed into a viscous mass similar to lye; sometimes, when they are of small dimensions, they become absorbed; the cavity which contains them contracts, and cicatrization is produced.

The hepatized lobules reappear but exceptionally in their primary condition after absorption of the exudation. They generally become atrophied, sclerosed, and atelectatic; they also frequently show lesions of caseous or fatty degeneration, of calcification or suppuration, of softening necrosis, or "putrid or cavernous transformation." In the pleura we observe callous or verrucose connective-tissue proliferations, which establish as a rule adhesions between the lungs and the pectoral walls.

These are the only important alterations met with in the lungs and the pleura. In the liver we may find a fibrinous interstitial alteration with atrophy of the hepatic cells. Sometimes we recognize sero-fibrinous exudations in the articulations, the synovial sheaths, the connective tissue of the fetlock, chest, etc. We may also find alterations of intestinal catarrh, arœolization of Peyer's patches, and ulcerations upon the gastro-intestinal mucous membrane (Degive).

Symptoms. After an incubation of from three to six weeks on an average (maximum, six weeks; minimum, a few days) the symptomatic evolution begins, which is usually divided into two periods:

1. The *period of development*, also called slow "chronic period" (*stadium occultum*), is marked in a general manner by the symptoms of a chronic pulmonary affection. Its average duration is from two to six weeks; it may, however, be reduced to a few days. Lobular inflammatory centres are located in the lungs. The first symptom is a short, painful, dry cough, which is very weak in the beginning, and is only heard in the morning, or when the animals rise, change their position, make exertions, or also when they have just been drinking; it becomes more and more marked and frequent; later it is extremely painful. When these spells occur the patients extend the head and neck convulsively and arch the spinal cord. We observe disturbances of the appetite and rumination; the mammary secretion is lessened. The fever is slight; the temperature varies between 39.5° and 40° C.; it is distributed irregularly upon the superficial regions. Percussion and auscultation do not reveal anything abnormal; we may hardly notice here and there a coarse vesicular murmur; in many animals the intercostal spaces are very sensitive to pressure. In some rare cases the process does not exceed this stage; its manifestations become gradually attenuated; then the cure follows.¹

2. The *acute period* is characterized by symptoms of serious pleuro-pneumonia and intense fever. In general, its duration is from two to three weeks, more rarely but a few days. Pulmonary phlegmasia progresses; it becomes lobar and diffuse. The respiration is much accelerated and laborious; the nostrils are dilated to excess; there is considerable beating of the flank. Most patients remain standing, with their hind legs spread, the knee-joints turned outside; if they take a decubital position, it is for a few moments only, and they lie constantly upon the diseased side. The cough is painful and very deep; the ribs and spinal cord are very sensitive to pressure. We observe a mucous nasal discharge, which is sometimes bloody—even purulent and fetid in some cases. In the beginning percussion gives a tympanitic sound; later it reveals a vast zone of dulness, which is limited above by a horizontal line. On auscultation we hear a weakened vesicular murmur; sometimes

¹ There are cases where the infected animals show nothing important during incubation. I have seen a bull which was brought into a stable three months before, and which appeared to be in perfect health; he was suddenly affected with intense fever. Within forty-eight hours two-thirds of his left lung were invaded. An examination of this organ showed no old lesion in it.—L. T.

it has been replaced by tubular blowing, râles, and friction bruits; in the healthy lung the vesicular murmur is strong and coarse. The other indications which are furnished by percussion and auscultation are the same as in pneumonia or a true pleurisy. Hyperthermia reaches 40° to 42° C.; the temperature is irregular in the various superficial regions; we count 80, 100, or more pulsations per minute. The ears and horns are sometimes cold, at other times hot; the muzzle is dry and hot and the legs cold, the hair bristly and thirst intense; the appetite, rumination, and lacteal secretion are entirely suppressed. We frequently observe constipation, which persists for several days. The urine sometimes contains a large quantity of albumin. In a few rare cases the beginning of the second period is marked by slight colics (stamping) and diarrhea. Most cows which are in calf abort. At a certain period emaciation makes considerable progress; œdema appears in the dependent regions (fetlock, chest, members), dyspnoea increases, the pulse becomes accelerated and weakened, the heart-impulse is palpitating. Soon the animals are no longer able to stand; they are found in complete lateral decubitus, and suffer pains which produce complaints and prolonged groans. Death occurs by asphyxia.

Course and prognosis. The course is sometimes acute; then, again, chronic. In general, it is rapid in young animals which are strong and well fed; in old animals which receive watery, non-alible food the disease has a slower course, but is more malignant; 30 to 50 per cent. of the affected animals die. Death occurs ordinarily from two to four weeks after the beginning of the acute period; in some cases, where the symptoms become rapidly alarming, it takes place within from five to eight days. Besides this mortality 30 per cent. of the affected subjects are imperfectly cured and remain affected by chronic lesions of the lungs. As a rule, pleuropneumonia kills or disables 50 to 70 per cent. of the patients. At least 50 per cent. are rendered absolutely useless. In a small percentage of cases the affection terminates in complete cure. The improvement begins toward the fifth day of the acute period; convalescence lasts for several weeks; the absorption of interstitial exudations proceeds very slowly. Sometimes the disease has an abortive course, and its symptoms are but slightly marked: slight cough, moderate acceleration of the respiration, etc.; the appetite is then generally preserved.

The persistent alterations of the lungs and pleura (necrosis,

cicatricial induration, connective-tissue proliferations and pleural adhesions) lead to permanent dyspnœa, cough, and progressive emaciation. The clinical aspect of chronic pleuro-pneumonia recalls that of pulmonary tuberculosis, by which it is frequently complicated. In this form of the affection we observe relapses after many months.

When the disease invades a crowded stable it at first attacks but few victims; but after several weeks it runs a very intense and increasing course. It may persist for months and become stationary.

The prognosis varies with the disease. In countries where pleuro-pneumonia has already penetrated and in those where it exists permanently it is always much less serious than in the regions where it makes its first appearance. Defective stabling and unsuitable regimen have an unfavorable influence upon its course.

Differential diagnosis. The diagnosis is very difficult in the initial period; the symptoms are then little marked and not characteristic.¹ Hyperthermia and cough become important when we have reason to believe in the existence of pleuro-pneumonia. Even during the acute stage the diagnosis *intra-vitam* is only possible in subjects which belong to an infected stable, or when several animals are affected simultaneously. As a general rule, it may only be established with assurance by the autopsy. From a differential diagnostic standpoint we must especially eliminate the following diseases:

1. *True croupous pneumonia.* This disease, which has an acute course and typic evolution, exists in a sporadic state; it is characterized anatomically by the uniformity of age of the lesions and by the non-participation of the interstitial connective tissue and pleura in the phlegmasic process (see Differential Diagnosis of Croupous Pneumonia of the Ox, vol. ii., page 235).²

2. *Tuberculosis.* At the period of development of pleuro-pneu-

¹ Tuberculosis is in fact extremely frequent in cows which have resisted pleuro-pneumonia. I saw it at the Vincennes farm previous to 1870, in most animals which were considered as cured. At that period they generally believed that the disease became transformed. It is hardly necessary to state now that tuberculosis constitutes a new morbid entity, which becomes implanted in a soil exceptionally prepared for it.—L. T.

² This affection never starts with such intense fever. In a working ox which I was able to observe well, I saw the fever increase with the lesion, while in pleuro-pneumonia it always precedes the appearance of typic symptoms, sometimes for two days. Finally the period of increase of sporadic pneumonia seems to be ordinarily accompanied by a rusty nasal discharge.—L. T.

monia it is often impossible to make a clinical differentiation of tuberculosis; in this latter disease, in fact, we also observe febrile attacks.

3. *Traumatic pneumonia or pneumonia by foreign bodies* may determine the same symptoms as pleuro-pneumonia. However, the manifestations of traumatic carditis, which quite frequently coexist with those of the pulmonary affection, permit us to establish a diagnosis.

4. *Epizootic disease of oxen (Rinderseuche)*. The pectoral form of this affection may also show great analogy with pleuro-pneumonia, and like this, it exists in an epizootic state. The distinction is based upon the simultaneous appearance of exanthematous and abdominal forms of the former, as well as upon its rapid course. Anatomically, the pulmonary localization of the *epizootic disease of oxen* is characterized by the uniformity of age of the lesions and by the acuity of the process (See *Epizootic Disease of Wild Animals and Oxen*).

5. *Verminous pneumonia and simple catarrh, pneumomycoses, acute pulmonary emphysema, pulmonary atelectasia*, etc., may also lead to errors.¹

The cases of foetal atelectasis, which are quite frequent in the calf, are interesting from a practical standpoint. They have been advanced in order to weaken the anatomical test of pleuro-pneumonia, which consists of a marbled-like disposition of the pulmonary tissue (hypertrophy of the interstitial connective-tissue tracts, hepatized islands of various age and coloration).

We may hope that in the near future the anatomical differential diagnosis will be rendered easy by a microscopical and bacteriological examination.

SEPTIC PLEURO-PNEUMONIA OF THE CALF. Under this denomination Pöls has described an epizootic affection of calves which offers a striking similarity to pleuro-pneumonia. It is found in different countries, and shows the stamp of a septicemia with localizations on the pleura and lungs. It is rapid in its evolution, and is accompanied by febrile symptoms and serious dyspnoea. The

¹ *Infectious broncho-pneumonia* of American oxen, described by Nocard, is produced by a short, ovoid and mobile bacterium found in the bronchial exudate, in the hepatic tissue, and in the slight amount of serum contained in the perilobular lymphatic pouches. From an anatomo-pathological standpoint this disease is especially distinguished from pleuro-pneumonia by the presence of muco-pus in the bronchi (Bull. Soc. vét., 1891).—N. D. T.

alterations of the lungs show a great similarity to those of pleuro-pneumonia. According to Pöls, the infectious agent is a bacillus similar to the bacteria of infectious pneumonia, septicemia of the rabbit, and epizootic disease of wild animals. In the pig the inoculation of cultures of this microbe produces a disease similar to infectious pneumonia; it is fatal for the mouse, rabbit, guinea-pig, calf, and young bovines.

Perroncito has studied infectious pneumonia of the calf, which occasions great damage in Italy. According to this author, it is said to be determined by a micrococcus (*Micrococcus ambratus*) which is easily developed upon gelatin and agar; it forms nail-shaped colonies and communicates a bituminous color to these substances. This disease is seen in calves up to the age of three months. It has also been observed in young pigs. In Russia it has been described by Semmer.¹

Treatment. All the medical agents formerly employed have proven inefficient. Pleuro-pneumonia must be combated by prophylaxis. The measures of sanitary police prescribed in German law in relation to contagious diseases (45 and 79), mainly in the slaughtering of diseased animals, give the most reliable results. In some countries pleuro-pneumonia is classified among diseases setting aside contracts of sale. In Bavaria the delay is forty days; in Saxony, thirty; in Baden, fourteen.

Inoculation. Inoculation has been tried and applied for a long time as a prophylactic agent. In the beginning of this century it was recommended in Germany by Haussmann and a few other veterinarians. According to Rochebrune, it is said to have been used in olden times by the Arabs upon the flocks of Senegambia. The operators introduce the point of a knife into the lungs of an animal which had died of pleuro-pneumonia, and afterward make, by means of this instrument, an incision into the skin of the nasal region.

But inoculation became very popular after the researches made

¹ Hutcheon has observed at the Cape of Good Hope a specific pleuro-pneumonia existing exclusively in subjects of the caprine species, introduced by angora goats. It is marked by symptoms of a serious pectoral affection, and terminates in death in two-thirds of the cases. It seems to be transmitted by direct contagion only. It has been produced experimentally by making healthy subjects ingest a certain quantity of pleural exudate. Preventive inoculation, when performed according to the rules established for pleuro-pneumonia, reduces the mortality to nearly 30 per cent. The duration of immunity varies from four to six months. (The Journ. of Comp. Med. and Vet. Archiv., 1890, and in Annal de Méd. Vét., 1890.)—N. D. T.

in Holland by Willems from 1850 to 1852. Since that period it has been studied in almost all countries, and numerous monographs have been published upon this subject. Nevertheless, it is very difficult even to-day to give a decisive opinion as to its value. We will detail the arguments which are advanced for and against it by its partisans and its adversaries.

The defenders of inoculation, among whom we must mention Haubner, Pütz, Rueff, H. Bouley, and Degive, base their opinion upon the fact that a first infection confers immunity for life. In support of their views they advance numerous considerations. By inoculation, say these authors, we start a specific inflammatory process in the caudal appendix which is of the same character as that which is developed in the lungs when pleuro-pneumonia is contracted naturally, and when the evolution of this local process is ended the organism is given immunity; besides, this operation shortens the duration of the epizootic in a stable, and the loss which it produces is very slight. Haubner estimates the number of these at 1 to 2 per cent., and those of cases of gangrene of the tail at 5 to 10 per cent. In inoculations made in Holland during the years 1878-79 the mortality was 0.66 per cent. (observation of 59,180 animals). In order to demonstrate the advantages of inoculation authors mention especially the results obtained in Holland (Spöling), in Saxony, and in Australia. Its advocates deny the transmission of the disease by inoculated animals; they point out that it is far less expensive than slaughtering, which leads to considerable loss; they contest finally the efficiency of the sanitary measures, even the most severe. According to one of Degive's statistics, out of 6708 animals inoculated in various countries from 1850 to 1863, the mortality was 2.7 per cent.; in 2453 animals which were not inoculated and exposed to contagion in the same conditions as the preceding, 26.9 per cent. contracted the disease. In Holland inoculation reduced the number of animals affected by pleuro-pneumonia from 6079 in 1871 to 2227 in 1875, to 951 in 1877, to 157 in 1879, and to 11 in 1882 (Pütz). At Hasselt, where it was practised with much success upon 200,000 animals in thirty years (1850 to 1880), an increase of loss was invariably observed during the periods it was neglected.

The adversaries of inoculation—among others Roloff, Wehenkel, Lydtin, Oemler, Zündel, Stiffen, Kitt, and Adam—oppose the following objections: The existence of complete immunity conferred

by inoculation can never be demonstrated. Its advocates are unable even to indicate in a precise way the duration of its prophylactic effects; some speak of incomplete immunity, and repeat the operation several times. We may doubt the specific influence of the local process which it determines, for a similar lesion may be produced by using pus or milk. By inoculation they have never produced pleuro-pulmonary lesions which constitute the test of pleuro-pneumonia, and which are so essential to it that they are even observed in the foetus in cases of intra-uterine transmission of the disease. The results of the inoculation depend upon the method employed, the time practised, and the quality of the virus. Inoculation was frequently performed upon animals which already possessed immunity, which was attributed to the effects of caudal inflammation. By inoculation disease is propagated and epizootics are perpetuated. The loss which it occasions is sometimes considerable; the mortality is often very high, sometimes exceeding that of pleuro-pneumonia. The commission which was intrusted with the framing of the German law upon contagious diseases has estimated the loss which would be caused by inoculation at 2 to 4 per cent. According to the observations of a French commission,¹ the number of cases of gangrene of the tail has been 25 per cent.; and according to one of Degive's statistics, from 10 to 15 per cent. The operation has drawbacks from an economical standpoint; it leads to a diminution of the mammary secretion, emaciation of the inoculated animals, etc. The epizootic often spreads by this means,² while in other cases, by not practising it, the disease dis-

¹ In experiments made by the Dumas commission in 1850 the proportion of subjects in which the gangrene of inoculation produced loss of the tail was 27 per cent., and the death-rate 11 per cent. (Report of H. Boulay: *Recueil Vét.*, 1854). But the percentages of these accidents are to-day much smaller. In 1354 animals inoculated by the commission instructed to combat pleuro-pneumonia in the department of Basses-Pyrénées in 1884, only 10 (less than 1 per cent.) died, 15 (1 per cent.) lost their tail, and in 30 (2 per cent.) this organ became partially mortified. (Report of Delamotte: *Bull. Soc. Cent. Vét.*, 1884.)—N. D. T.

² A few observations have been mentioned which appear to establish, on the one hand, that inoculation may produce pleuro-pneumonia (Degive, Mollereau, Willems, etc.), and on the other that the inoculated animals may communicate the disease to others (Reynal, Cagny, etc.). But the subjects in which this became developed under these circumstances had certainly been contaminated previously. Caudal inoculation performed upon animals free from all virulent impregnation has never produced pleuro-pneumonia with its typic pleuro-pulmonary lesions. Not one authentic fact has ever been advanced demonstrating that preventive inoculation had created an infectious centre or a starting-point of an epizootic of pleuro-pneumonia.—N. D. T.

appears of itself. Many animals contract this affection and recover unobserved. Pleuro-pneumonia does not disappear in countries where inoculation is most practised (Belgium); in others, like Holland, the extinction of this plague has not been the result of inoculation, but of the strict application of other measures of sanitary police, especially slaughtering. In Belgium, according to Oemler, the number of cases of pleuro pneumonia increased from 1481 in 1867 to 2800 in 1878, notwithstanding that inoculation was practised regularly during this entire period; this number afterward considerably diminished through sanitary regulations: in 1880 it was 1781; in 1883, 1187. According to a statistic of Kitt, the number of cases of pleuro-pneumonia in England was 4590 in 1878; by strict enforcement of sanitary measures it was reduced to 2144 in 1879, and to 1200 in 1882. In Bavaria, since the promulgation of the law on contagious diseases, the number of cases of pleuro-pneumonia has fallen from 846 in 1846 to 281 in 1883. In Baden, where the annual loss was 0.2 per 1000 from 1870 to 1880, no case has been observed since 1885.

In considering in an impartial manner the arguments for and against inoculation, it is difficult to form a definite opinion as to the value of the method, because the experiments and observations are as yet not sufficiently numerous. At the present time it does not seem indicated to recommend *preventive inoculation*,¹ but only *inoculation of necessity*, in order to shorten the duration of the epizootic in an infected stable. We consider the measures of sanitary police, especially slaughtering, as the most efficient means to combat pleuro-pneumonia; this affection belongs to the group of contagions, the agent of which is not very widespread, and the propagation of which may be arrested by slaughtering the animals. From a prophylactic standpoint this measure offers much greater chances of success than inoculation. The preserving virtue of this latter is yet problematic. At all events it is very desirable that

¹ Inoculation as a means of precaution is nevertheless indicated in countries where many cattle are imported and in which pleuro-pneumonia exists permanently. In Holland it is used in the distilling regions; from 1878 to 1882 they inoculated 128,360 animals with an average loss a little below 1 per cent. In the animals which were not inoculated the mortality was 26 per cent. As is pointed out by Sanson, while the Hollandish veterinarians for a long time discussed the advantages and drawbacks of inoculation, the breeders were unanimous on the question of its value; they knew that without inoculation their industry was impossible.—N. D. T.

bacteriological researches may soon give us a more perfect and scientific process of inoculation.¹

The practice of inoculation varies with the method employed. The injection is mostly made upon the extremity of the tail. In order to obtain the virulent lymph we select an animal in which the disease exists in its primary stage; we incise hepatized fragments of the lungs, which should be slightly compressed to extract the serum; this fluid is then allowed to coagulate, and is afterward filtered through a piece of fine linen. Some practitioners use the liquid obtained by incisions in the caudal tumefaction; the lymph thus obtained is said to be less virulent.

Inoculation is performed by means of Sticker's needle or a canulated lancet; the selected region is the dorsal fascia of the tail at a distance of eight to ten centimetres from the extremity of the organ. After having cut the hair upon this region, we make one or two virulent punctures sufficiently distant from one another and penetrating as far as the subcutaneous connective tissue (subcutaneous inoculation). The lymph must be limpid and of yellowish-white color; it is essential to draw it from pulmonary tissue which is not much altered, hepatized and free from necrotic centres. We obtain positive results in 75 to 90 per cent. of animals. Within one to four weeks the region where the liquid has been inserted becomes the seat of an inflammatory tumefaction which does not ordinarily

¹ Every day new facts are demonstrated which testify to the efficiency of preventive inoculation, and the proof that it truly confers immunity to animals subjected to it is irrefutable on account of the sterility of the virus in a *protected region*. While in animals free from any infection the operation practised upon any other region than the caudal extremity is followed by intense inflammatory and gangrenous accidents, which are often fatal in inoculated animals, it remains without effect—it produces only insignificant phenomena. This is the test of immunity which is acquired by inoculation. In order to obtain good results from it it is indispensable that it be practised as soon as the disease appears in a stable or as near as possible to the beginning of the disease. If it is made at a late period, a certain number of animals are already contaminated; in these pleuro-pneumonia is in process of evolution, and inoculation cannot stop its development. Leblanc has, however, opposed facts which are of great interest in relation to hasty inoculation. In 1882, in 14 stables, and in 1883, in 23, before practising the operation, he thought it proper, on account of the peculiar circumstances, to wait for a case of pleuro-pneumonia, and this second case did not happen!—N. D. T.

[That inoculation for contagious pleuro-pneumonia is a failure is proven by the fact that it exists to this day in all countries where it has been systematically practised. The only countries where this disease has been eradicated are those where sanitary police measures, especially slaughtering, were enforced. This method has completely exterminated the disease in the United States, in which there has not been a single case for over three years.—W. L. Z.]

exceed the dimensions of a hen's egg. We then observe a low fever and a slight acceleration of the respiration. When impure lymph has been used an extended tumefaction is developed upon the entire length of the tail, the extremity of the organ becomes necrotic, the fever intense, then symptoms of septicemia and pyemia appear. The same phenomena occur when inoculation is practised upon protected regions, particularly upon the fetlock or the root of the tail.¹

Serious inflammatory processes developed in the tail may end fatally; they are relieved by free incisions, by local antiseptic treatment, and by amputation of the necrotic part. The average mortality is about 1 to 3 per cent.; 5 to 15 per cent. of the patients lose their tails.

Sometimes within six or eight weeks a second inoculation is practised above the point where the first was made.

Martin makes the inoculation by introducing under the skin, on the surface of the last coccygeal vertebra, a small seton three millimetres wide dipped into lymph. Rutherford has obtained good results by this method; the mortality was 2 per cent.; serious accidents were much more rare than when using the ordinary process. The tumor of inoculation appeared from the ninth to the fourteenth day; it remained prominent from seven to nine days, and disappeared within fourteen to twenty days.

Thiernesse, Defays, H. Bouley, Degive, Sanderson, and others have practised intravenous inoculation by injecting directly into the jugular vein 2 c.c. of lymph. As a rule, the results have been excellent. In one case a pneumonia was developed at the autopsy, the lungs showed on section the characteristic marbled aspect of pleuro-pneumonia.

¹ In experiments which Pasteur made at the farm of Vincennes (1882) he recognized that gangrenous accidents of the caudal extremity had to be ascribed exclusively to the specific action of the virus.

In 1889 and 1890 Schütz and Steffen made researches upon the inoculation of pleuro-pneumonia practised by using all the antiseptic precautions (shaved skin, soaping of the region and washing with a sublimate solution, sterilization of instruments, disinfection of the hands, etc.). The principal conclusions of the work are:

The lymph of pleuro-pneumonia which is yet warm is more virulent than cold lymph and the hepatized pulmonary tissue. Immunity is much more certain when the local phlegmasia produced by inoculation is marked. The extension of local processes is not at all in proportion to the quantity of virus used. Inoculations made with lymph still warm, even when applying antiseptic precautions, is not always without danger. Inoculation of fresh lymph determines in the organism of the ox generating phenomena of immunity. This is truly given by inoculation (Berlin Archiv, 1889-90).—N. D. T.

DOURINE: EQUINE SYPHILIS.

HISTORY. This disorder is designated under the name of *venereal disease, disease of coitus, paralysis of breeding animals*. Dourine was observed for the first time by Aunmon in the studs of Tra-kehnen in 1796. In 1817 it appeared in Hanover (in the studs of Celle), in Schlesien, and in Poland, where it occasioned great losses from 1830 to 1840. In 1840 the Prussian Government prescribed sanitary measures in order to combat it. About 1821 it penetrated into Austria, especially in Hungary and Bohemia; it became widely extended from 1859 to 1862. In 1830 it was introduced into Switzerland, where it made great inroads in the stud farms; in 1836 it invaded Italy, in 1843 Russia, and later Algiers, Syria, etc. According to Röhl, it did not invade England or Belgium. Formerly it produced enormous losses in Germany, but has almost entirely disappeared from that country since the promulgation of the law on contagious diseases; of late years the official sanitary reports of the German States have not mentioned a case of it.

Various hypotheses have been advanced as to its nature. According to the first, which is the most widely accepted, dourine is identical with syphilis of man, whence come the denominations *venereal disease of the horse, chancre disease*, which have been given to it. Quite recently also Bouley, Trasbot, Laquerrière, and others have admitted that dourine was but syphilis, which had first been transmitted by syphilitic Arabs to the female donkey, then by this to the stallion donkey, and this in its turn communicating it to the mare. Laquerrière has advised to give to it the name of *equine syphilis*. But this doctrine is not based upon any positive fact. Other authors see in dourine a kind of glanders of the genital organs; they assert that in some cases it may be transformed into glanders or farcy. Finally, a few veterinarians have maintained that it consists essentially of a true affection of the spinal cord, which is specially marked by a paralytic phenomena. They have named it *paralytic disease* (Strauss), *nervous disease, paralysis of breeding animals*; this latter appellation is frequently used nowadays.

We have long confounded under the name of dourine two pathological states, which are radically different: *true dourine* and *vesicular eruption* of the genital organs (*coital exanthema*), which

has much obscured the nature of the disease. Most ancient descriptions are so confused that it may be very difficult in reading them to recognize with which of these two affections the observers have had to deal. It is only of late that they distinguished in disease of coitus a benign and malignant form, corresponding with vesicular eruption and true dourine (Hertwig). ~

Even to-day these two morbid species are imperfectly known as far as their etiology and symptomatology are concerned. Besides Thanhoffer's works no clinical researches have been made upon their precise anatomy or bacteriology.

Etiology. Dourine is a chronic infectious disease, which at first is localized upon the genital mucous membrane, and which becomes more or less rapidly generalized. It is particularly observed at the periods of copulation; it is transmitted exclusively by coitus; it is especially propagated upon the stallions of the government stations and the *migrating stallions*; the mare contributes much less to its dissemination. All the mares which are served by a stallion affected by dourine may be contaminated. On account of the chronic course of the process the affected subjects are contagious for more than a year. According to Thanhoffer, the infectious agent is a micrococcus which exists in the vaginal secretions, in the sperm, the spinal liquid, the medullary roots of the peripheric nerves (ischiatric), and in the blood. According to Hertwig, the disease is transmissible by inoculation of the genital mucous secretions; the blood, sperm and prostatic liquid, etc., have not appeared virulent. Transfusion of blood has given negative results to Trasbot and Saint-Cyr. According to Roloff, Jessen, and a few others, dourine is hereditary.¹

Pathological anatomy. The lesions of the genital apparatus consist essentially in an œdematous or phlegimonous tumefaction of the vulva and of the neighboring regions in the mare; of the penis, sheath, and scrotum in the entire horse; when the disease is old the skin of the sheath is often thickened and indurated (adipose sheath). Upon the mucous membrane of the lips of the vulva, upon those of the vagina, urethra, and surface of the penis, we observe symptoms of catarrhal inflammation, a thickening of the integument,

¹ In some rare cases dourine may be transmitted by mediate contagion. Blaise has recognized that it may be inoculated with the lancet. Subcutaneous injections made with blood coming from infected horses have given him positive results in three mares.—N. D. T.

ulcerations and cicatrices; these ulcerations are sometimes superficial, in other cases deep; the cicatrices are smooth, white, hard, and callous.¹ The vaginal mucous membrane is affected with circumscribed, pustular or villous proliferations. Upon the vulva and penis we observe discolored spots which have been produced by ulcerations (toad spots). The testicles, which are hypertrophied or atrophied, contain caseous inflammatory centres. In the vaginal sheath we find fibrous adhesions and villous proliferations; the connective tissue of the epididymis and of the testicular cord is infiltrated by a yellowish gelatiniform exudate. The mammæ are sometimes inflamed, tumefied, and permeated by small abscesses.

In the spinal cord Thanhoffer has recognized lesions of myelomycosis: a central hemorrhagic myelitis, or a syringomyelitis (central medullary sclerosis), or localized degenerative processes. Upon certain more or less extensive regions the marrow is thinned; on section it appears asymmetric, softened, dotted red; the gray substance is atrophied and contains small, softened centres. At times the meninges are dull, adherent and fast in some places; sometimes they are hyperemic, and we find in them a variable quantity of serous exudate. The encephalic meninges offer similar alterations; the cerebral substance is often cedematous and the ventricles contain a considerable quantity of serous liquid; the intermuscular connective tissue and the neurilemma of the great rhachidean nerves are the seat of a gelatiniform infiltration. The microscopical examination reveals alterations of the ganglionic cells (nuclei and bodies), of the cylindrar, neuroglia (the quantity of which is increased), and of the bloodvessels.

As a rule, the lymphatic ganglions of the genital organs are tumefied and pigmented, and contain caseous centres varying in size from that of a pea to a nut. In the abdominal cavity the subperitoneal tissues are infiltrated and the mesenteric ganglions are sometimes the size of an apple. Ruthe has observed on the intestinal mucous membrane rounded ulcerations. The skin is marked with circumscribed blotches of variable dimensions, quite hard, and produced by serous infiltration of the derma (vaso-neurosis). The respiratory mucous membrane shows, in some points, catarrhal lesions. We also observe considerable emaciation and all the symp-

¹ It is rare, especially in stallions, to observe chancres or cicatrices of chancres. Blaise has not seen these lesions upon any of the twelve patients of which he has related the history in his work.—N. D. T.

toms of anemia. In some patients we find hypostatic pneumonia. When dourine is complicated by pyemia we find metastatic centres in the lungs.

Symptoms. The clinical tableau varies with the period of the disease. In the beginning this is marked by phlegmasia and ulcerations of the genital mucous membrane. When the general affection is complete it determines a medullary affection which is marked by spinal paralysis and cutaneous vaso-neurosis (urticaria).

1. The primary alterations of the genital organs appear after a period of incubation which, according to Marech, varies from eight days to two months. In the stallion the first symptoms are tumefaction of the penis, especially of the glans; we sometimes observe paraphimosis the same as after castration. Upon the penis we find red spots, vesicles, and ulcerations. The orifice of the urethral canal is red and tumefied and allows the passage of a mucous exudate. The patients place themselves frequently in the same position as when urinating; we also frequently observe an abnormal genesic ardor. The inflammation may be propagated to the sheath, serotum, and testicles (orchitis). Later, the lymphatic vessels and inguinal ganglions are invaded. These local lesions may disappear almost entirely in the course of the disease; they are wanting in cases where the process evolves upon the urethral mucous membrane; we observe then violent efforts at micturition, and the urethra discharges a mucopurulent liquid.

In the mare dourine begins by cedematous obstruction of the vulva, which may extend to the mammae and external fascia of the thighs. The vaginal mucous membrane is spotted red and tumefied; it sometimes forms thick rugæ and becomes covered with a turbid, yellowish-red matter, where it is strewn with pimples, vesicles, and ulcerations; but in the mare the local lesions are often insignificant. The redness in the neighborhood of the clitoris is most marked; this organ is inflamed and turgescens. The genesic excitement is much marked; the patients, which are very ticklish, stretch themselves constantly and make violent expulsive efforts in ejecting small quantities of urine; in some cases a viscid, grayish mucus is expelled; the tail is wagged constantly; the vulva is open and shows the erect clitoris.

Sometimes the vulvar discharge is ichorous and irritating; it soils the tail and hind legs and produces excoriations. In serious cases the lymphatic ganglions of the pelvis are tumefied, as are also

the mammae which may present abscesses in several points. The tumefaction sometimes extends to the lower regions of the abdomen.

2. The general symptoms follow weeks or even months after the local accidents, which by that time have mostly disappeared. The patients are very weak, the posterior members are dragged while walking; we also observe frequent spasmodic flexions of the fetlock.

At this period we observe upon the surface of the skin, especially in the stallion, round blotches, which may be as large or larger than a silver dollar; they are distinctly circumscribed, and may reach the uniform thickness of a finger; they are produced by a serous infiltration of the papillary body of the derma in regions which correspond to small cutaneous arterioles; they seem to be connected with vascular neuroses. These blotches are often developed very rapidly and in some instances disappear just as quickly. In most cases, however, they persist for several weeks, and gradually become harder and slowly resolved. They are usually found upon the withers, neck, and shoulders, chest and abdomen.

Soon we see progressive paralysis of the hind quarters and considerable emaciation. The walk is uncertain and staggering; we recognize spasmodic flexions of the fetlock and metatarsus (stringhalt) of the hind legs;¹ some patients fall frequently and have difficulty in rising; others are affected by general trembling or by paralysis of the lips, ears, and eyelids (ptosis); erection and rearing being impossible, the stallion cannot perform service. In some cases, but particularly in the males, we may find upon the skin a remarkable hyperplasia, which is marked by intense pruritus; the patients scratch and rub themselves constantly; the derma is exposed on large surfaces. Emaciation is marked and the hind quarters become skeleton-like. The skin is dry, the hair dull and bristly. The lumbar region is the seat of great sensitiveness. The general state becomes aggravated, apathy is more and more marked; the eye is haggard and without expression. Finally, the animals remain constantly down, and perish of hypostatic pneumonia or of septicemia consecutive to the lesions produced by decubitus or cachexia. The appetite remains normal for some time. Laguerrière has observed serious internal ophthalmia. Toward the last we

¹ We also see at times transitory paralysis of a foreleg. As a general symptom we ordinarily find a lowering of temperature below the normal in the secondary period.

—L. T.

may sometimes see nasal catarrh, conjunctivitis, and tumefaction of the lymphatic ganglions of the submaxillary.

Course and Prognosis. The course of dourine is always chronic. Its duration is from six to twelve months, but it may last two, three, or four years, and even longer. The abortive form, in which the process consists of local lesions only, is extremely rare. It is common to see relapses after marked improvements. In general, the local symptoms become attenuated, and disappear at the same time as the general infection takes place.

The prognosis is most obscure. The mortality is 70 per cent. A cure is only possible in the beginning. In the stallion the disease is more serious than in the mare.¹

Differential diagnosis. The diagnosis is ordinarily very difficult for quite a long period, until the time where the pathognomonic symptoms (urticaria and paralysis of the spine) appear. It is especially uncertain for the stallion, because in him the genital organs do not usually present any visible lesion; the affection has often only been recognized when several mares had already been contaminated.

It is probable that dourine has frequently been confounded with coital exanthema, a benign affection of the genital organs, which is marked by local lesions objectively similar to those of dourine, but not accompanied by any general disorder. The ulcerations and inflammation of the genital mucous membrane may be mistaken for glanderous lesions, when we see at the same time lymphangitis and adenitis. The course of the disease and inoculation permit a differentiation. In the mare and entire horse affected by "fetid discharge" we have sometimes observed, after coitus, phlegmonous pyogenic processes, with tumefaction of the lymphatic ganglions; these processes present a certain similarity to those of dourine; the ultimate course gives us also the necessary information in this case. Sometimes, finally, the locomotor troubles of chronic paraplegia simulate those of the second period of the disease of copulation.

Treatment. The treatment may only be efficient in the begin-

¹ In 1886, of 26 stallions affected by dourine at the station of Blidah, 16 died, 4 were sold after castration, and 6 appeared to have recovered for a time (Blaise). According to Laguerrière, recovery is very rarely complete. Often, after two or three years, animals supposed to have been cured, are attacked with nervous, cerebral or medullary accidents, and die.—N. D. T.

ning. We must then combat the local lesions by disinfectants (solution of sublimate, phenicated water, etc.). When infection has taken place the local treatment by disinfectants and astringents (bran and water, alum, tannin, sulphate of iron, etc.) is purely symptomatic. Cutaneous tumefactions upon the mammæ and the lymphatics require surgical means: incisions, vesicant applications, etc. Internally, it is claimed we may use with success the medicinal agents which are generally directed against human syphilis (1 to 2 grammes of sublimate, 10 to 20 grammes of iodide of potassium), and also arsenious acid in small doses (0.5 to 1 gramme). In cases of spinal paralysis, resort to stimulating liniments. In the stallion authors claim to have cured dourine by castration; in order to explain this result they declare that the infectious agent is sometimes located exclusively in the testicles.¹

The measures of sanitary police enforced in the German Empire are indicated in articles 50 and 51 of the law upon contagious diseases, and in articles 110 to 116 of the instruction which completes said law.

INOCULABILITY OF SYPHILIS FROM MAN TO ANIMALS is not as yet established in a definite way. Positive results have, however, been claimed, especially in the monkey. We quote below the principal authors who have practised inoculation with success:

1. In the rabbit: Auzias, Turenne, Gailleton, Waller, Klebs, etc.
2. In the guinea-pig: Legros, Bradley, Michot.
3. In the cat: Auzias, Turenne, Bradley, Diday, Lyons school.²
4. In the dog: Auzias, Turenne.
5. In the horse: Mathieu.
6. In the pig: Martineau, Hammonie.

We may oppose these positive results with a series of negative facts obtained by Horaud and Peuch (dog, cat, mule), Hunter, Ricord, Horaud and Cornevin, Teleschinski (pig), Wertheim, Jessen, Tissoni (rabbit, dog, cat), Hunter, Depaul, Reynal (ox), Lacroix and Danet (ox, rabbit), Rebatel (dog, guinea-pig, rabbit), Letnick (pig, rabbit), and others.

Syphilis of animals possesses a certain interest from the stand-

¹ Instead of producing a cure castration almost always produces a marked aggravation of the trouble, especially cutaneous lesions, and sometimes death.—N. D. T.

² Journ. de Lyons, 1851.

point of comparative pathology ; but contamination of animals by man has never been observed.

COITAL EXANTHEMA OF HORSE AND OX.

I. Coital Exanthema of the Horse.

Etiology. Coital exanthema of the horse is a benign vesicular eruption, localized upon the vaginal mucous membrane and on the skin of the vulva, or upon the integument of the penis. It is a contagious disease with typic course. It has also been designated as *aphthous eruption*, *phlyctenular eruption*, *coital eruption*. Although this affection should have been described in the chapter on acute exanthema, we treat it here because it is regulated by sanitary measures.

It is observed upon several species, but particularly in the horse and the ox ; it is less frequent in the sheep, goat, or pig. The infectious agent is unknown. Our experiments have demonstrated that contagion is very subtle ; it takes place, as a rule, through coitus, but it is not rare to find contamination from mare to mare without the intermediation of copulation. In the suckling colt infection may take place by the lips, buccal, nasal, and ocular mucous membranes, and upon the posterior legs, by the morbid secretions of the mother (Dayot, Lundberg, Hess, etc.). The contagious germ is located exclusively in the contents of the vesicles or pustules and in the liquid secreted by the ulcerations. When a sick stallion contaminates a large number of mares—which is quite common—the affection assumes an epizootic character. *A first infection does not create immunity. The stud may become infected a few weeks after recovery from the first eruption* (Steinhoff).¹ Ex-

¹ In 1880 Peuch studied, in several localities of Haute-Garonne, an epizootic coital exanthema which existed in solipedes, and had the appearance of dourine. He recognized that he had simply to deal with *horsepox*. Inoculations made upon a cow and a heifer around the vulva, with the product obtained by crushing in a little distilled water scabs obtained from dried-up pustules, resulted in an eruption of true *cowpox* ; for the inoculation in man of the serum of the pustules developed upon these two subjects produced *vaccinia* (Revue Vét., 1880). In 1887, in the Haute-Loire Galtier also observed upon the horse an enzootic of coital exanthema which simulated dourine. By inoculation in calves he has determined *cowpox* with its typic characters (Journ. de Lyon, 1887). (See H. Bouley : Le Progrès en Médecine par l'Experimentation ; Leçons sur Horsepox, Cowpox, la Vaccine et la Variole, Paris, 1882.)—N. D. T.

anthema is sometimes communicated to man, mostly to stablemen. Inoculation usually takes place upon the hands; a varioliform eruption is produced, sometimes a swelling of the entire obstruction of the arm, tumefaction of the axillary ganglions, and an elevation of temperature. These phenomena always disappear rapidly.

The spontaneous development of the vesicles of coital exanthema can not be accepted. The "feverish eruption," which is looked upon by Haubner as a variety of vesicular eruption (coital exanthema), is a true eczema, which is not at all infectious.

Symptoms. 1. In the ordinary benign form the period of incubation is from three to six days; sometimes it is even shorter for the mare. In this animal the eruption is determined upon the vagino-vulvar mucous membrane; this membrane, which is hyperemic, shows dark spots of the size of a pin's head, upon the surface of which we soon see pimples, vesicles, and pustules, varying in size from a lentil to a pea, and having a very thin covering and limpid, reddish-yellow contents. The vesicles are especially numerous in the neighborhood of the clitoris; they may be seen in the external integument of the vulva and the lower fascia of the tail; they become transformed into superficial ulcerations with red ground, and produce a lymphoid, viscous, yellowish liquid; sometimes they become confluent and constitute large wounds covered with a brownish crust. There are, besides pruritus, great genesic excitement, attempts at micturition, and more or less intense vaginitis. The general symptoms (fever, etc.) are wanting. In the place of ulcerations we find smooth, round, whitish cicatrices.

In the stallion the penis is tumefied, red, and covered with "pimples," vesicles, and pustules, especially upon the surface of the gland. These lesions are soon transformed into more or less deep ulcerations, which heal and leave non-pigmented cicatrices. More rarely the eruption extends to the sheath and pouches; Schleg has observed it upon the inner fascia of the four legs and upon the neck; it had been developed after a coitus and by a deposit of morbid secretions upon these regions, coming from a mare. In many cases we may observe a urethral discharge; the patients have frequent erections and take the same position as when urinating.

Recovery generally occurs within three to four weeks without the help of any medication.

2. In the serious form the symptoms are more marked. In the mare the tumefaction of the vulva may become radiated to some

extent upon the posterior legs (as far as the hocks), and affect the chest. The lymphatic vessels, as well as the corresponding ganglions, participate in the inflammation, and may form abscess. Purulent centres are developed upon the mammæ, in the neighborhood of the anus, and at the base of the tail. The ulcers are deep and their secretion ichorous; at times they extend to the uterine mucous membrane. We observe, besides, alarming general symptoms and intense fever; the gait is stiff, and the emaciation considerable. Its duration is long (six months and over). After the resolution of inflammatory tumefactions we often see an obstinate chronic vaginitis. In some isolated cases the disease ends in death (Steinhoff). In the stallion we find also this serious form, which is marked by tumefaction and abscess formation of the inguinal ganglions, by fever, digestive trouble, lameness, and by the length and tenacity of the process.

In many cases these accidents have led to confusion between coital exanthema and dourine. They may be explained only by admitting septic or pyemic infection. Their treatment comprises the same indications as that of dourine (antiseptic treatment, incisions, etc.).

The benign form of coital exanthema does not require any special medication. We may, however, resort to antiseptic and astringent detersions (phenicated water, solutions of sublimate, alum, etc.).

II. Coital Exanthema of the Ox.

Etiology. Coital exanthema is more frequent in the ox than in the horse. In Prussia, during the years 1884-85, it was observed in 784 animals of the bovine species and in only 75 horses. In 1886, in the various provinces of Germany, it was recognized in 3944 oxen and 248 horses; in Bavaria, during the year 1885, it affected 455 oxen and 9 horses; and in Würtemberg, 362 oxen and 13 horses. This frequency of the affection in bovines is but relative; it is due to the fact that the number of these animals is much greater than the subjects of the equine species.

In the ox also the eruption has a very subtile contagiousness. All the cows of one village may be contaminated by the same bull. The cases of contagion outside of copulation are much more frequent in the horse. The disease has at times been transmitted to cows by the intermediation of a sponge which had been used in

washing the affected animals. Dinter has seen it propagated from one affected cow to nine others: contamination was produced by the friction of diseased animals against healthy ones and by continual wagging of the tail. In oxen Numann has observed the eruption in the neighborhood of the anus and sheath. According to Armbruster, contagion frequently takes place by wooden drains, which are used for the discharge of lochia (when the animals lie down the genital organs become affected by coming in contact with these drains). According to Kampmann, it also takes place through the air.

The disease may affect the same subjects several times. It has been observed three times in bulls at short intervals (Schneiper and others).

Symptoms. In the cow we see at first the symptoms of serious vaginitis. The vagino-vulvar mucous membrane is red, tumefied, covered with dark-red spots and coated with muco-albuminous secretions. We observe, besides, vesicles and yellowish translucent pustules varying in size from a millet-seed to a pea, and which become rapidly transformed into ulcerations; the exudate then becomes purulent, soils the posterior members and the tail, and dries up forming crusts. The ulcerations are often accompanied by local induration; their borders are dented and thick; their base has a bad aspect; in these cases they leave white cicatrices, which are smooth, regular, or star-shaped. A manual exploration of the vulva produces intense pain. The genital organs are the seat of a more or less intense pruritus. The patients are very irritable and ticklish in the region of the hind-quarters. Micturition is difficult and always preceded by violent efforts. Sometimes we observe abnormal genesic excitement. The course is often rapid.

When the ulcerations are very numerous the exudate is abundant, thick, irritating, and putrid; it determines excoriations in the neighborhood of the vulva and upon the posterior members. In some serious cases the mucous membrane shows gangrenous islands, the elimination of which is followed by losses of substance which vary in size from a nut to a hen's egg; this complication occurs when the infection has taken place through a wound. In a majority of patients we observe a passing hyperthermia; the appetite, rumination and lacteal secretion are temporarily suppressed. We find quite frequently chronic vaginal catarrh. Exceptionally the females abort.

In the bull the gland, sheath, and scrotum are inflamed, tumefied, and very sensitive to the touch. The penis is studded with "pimples" varying in size from a juniper-berry to a pea, also with vesicles and ulcerations; from the urethral orifice escapes a yellowish purulent mucus. Micturition is frequent and preceded by violent expulsive efforts; the quantity of urine expelled is small; phimosis is common. At the time of erection the ulcerations often bleed. The animals are weak, slightly constipated; the appetite is lessened, and rumination suppressed. The penis becomes gangrenous on large surfaces and deformed in exceptional cases (Kampmann). The duration of the affection varies from eight to fifteen days, more rarely three to four weeks. The epizootic may prevail from two to four months in the same stable. Coital exanthema must especially be differentiated from aphthous fever and cowpox. It is easily distinguished from bovine plague.

Concerning the treatment of coital exanthema, we should watch the progress of the disease, and not interfere with therapeutic measures unless warranted by special symptoms. In the serious form we must resort to disinfecting and astringent lotions (phenicated water, cresol, alum solution, sulphate of iron, etc.).

BACTERIDIAN ANTHRAX. MALIGNANT PUSTULE.

HISTORY. Bacteridian anthrax is the most ancient infectious disease of animals that is known. Moses, in his second book (v. 9), mentions it and calls it the "sixth plague of Egypt"; in his third book he indicates the transmission of anthrax to man by the intermediation of soiled clothing. The epizootic disease described by Homer in the first book of the *Iliad*, and which affected man, mule, and dog, was probably no other than anthrax. In the ninth book of his *Metamorphoses* Ovid gives an exact description of anthrax epizootics. Plutarch has given the history of an epizootic of anthrax which existed in Rome about the year 740 before our era. Dionysius, of Halicarnassus (488 before Christ), and Livius (425 before Christ), have mentioned examples of anthrax affections which existed first in animals living upon pastures, then in such as were kept in stables, in animals destined for sacrifices, in priests, shepherds, farmers, and lastly upon the whole population. Lucretia (428 before Christ) designated anthrax by the term *ignis sacer*; Columella has given to it the name of *malignant pustule*. Virgil has

described a disease of the sheep which was transmitted to man by the contact of furs, wool, and cadavers, and produced in the integument ulcerating lesions which extended to the muscles. Pliny speaks of a carbuncular affection which existed in Gaul (old France) in the neighborhood of Narbonne. The Arabian physicians knew anthrax under the name of *Persian fire*. Mezeray (966 after Christ) called it *St. Anthony's fire*. In Italy John Wierus has mentioned several epizootics of it in the second half of the sixteenth century (1552, 1598, 1599). At this period the senate of Venice prohibited, under penalty of capital punishment, the offering for sale any meat coming from cattle affected with anthrax. In 1617 Athanasius Kirchner gave the description of a disease of the bovine species which was transmissible to man and which killed 60,000 people. In 1662 gloss-anthrax became much extended in the neighborhood of Lyons. From 1710 to 1713 it spread all through France. Ramazzin observed an epizootic of it in oxen and pigs in the neighborhood of Padua. In this form anthrax has existed with intensity in almost all domestic animals (horse, donkey, ox, sheep, pig, stag, dog, chicken(?), fish(?)), also in man, and in most countries: in Germany (at first in the neighborhood of Augsburg), and in Hungary (1712), in Poland, Silesia, and Saxony (1726), in France (1731-1757). It invaded Franconia in 1755-61, Finland and Russia in 1758-59, Guadeloupe in 1774. Chabert (1780) has shown that the different forms of anthrax constitute one and the same disease; the division established by this author and the denominations which he adopted have been preserved up to date. In 1805 Kaüsch gave a good description of it, but he did not recognize its contagiousness. Soon after that period extensive epizootics of anthrax were observed (1807, 1810, 1819, 1827). From that time a sensible decrease took place in the losses produced by this plague. Anthrax of the sheep was well studied later by Delafond and Gerlach (1845). While Delafond did not believe in contagiousness, Gerlach demonstrated it experimentally (1845). In 1850 Heussinger published a work upon anthrax which was remarkable from a historical and geographical standpoint. He considered it as a *malarial neurosis*.

In 1855 Pollender (Wipperfürth) announced that in 1849 he had found in the blood of bovines affected by anthrax a considerable quantity of fine little sticks. These were also seen by Davaine (Paris) in 1850, and by Branell (Dorpat) in 1857. This last

author, who had seen these little sticks in the blood during life, based the diagnosis and prognosis of the disease upon their presence; but he absolutely denied their pathogenic properties. It was in 1863 that Davaine recognized that these elements were bacteria and that they constituted the specific agents of anthrax. At that period H. Bouley Sanson and many other veterinarians did not share Davaine's opinion. Cohn was the first who considered the little sticks bacilli and suspected their sporulation. R. Koch has enlightened us upon the development of spores and their transformation into the bacilli. He has also cultivated the bacteridium and has made a biological study of it.¹

Preventive inoculation, which was discovered by Toussaint, and then studied by Pasteur, Chauveau, etc., offers much theoretical and practical interest.

Bacteriology. Anthrax is determined by a sporogenous bacillary Schizomycete, the *Bacillus anthracis* or anthrax bacteridium.

1. The *bacteridia* appear in the living organism in the form of straight rods, which are cylindroid and immobile, the extremities of which are distinctly marked and slightly embossed; their average length is 5.7μ ; their width about 1μ . Their dimensions vary considerably according to the species of animal in which they are found and also according to the individuals; they oscillate between 1 and 14μ ; in general, their length is nearly double the diameter of the red blood-corpuscle. They are larger than most other bacteria. They are often found bent in the middle, and in some instances undulated upon the borders. If examined without staining, they show a homogeneous and uniform structure; their fundamental substance, which has the transparency of glass, is, according to Nencke, "anthrax-proteine"—that is to say, a kind of albumin which is non-identical with mycoproteine of other microbes. The stained bacteridium appears composed of several joints of a length somewhat less than the diameter of a red globule and separated by narrow transverse bands; the extremities of these elements, which are larger than the body, are marked by a central depression in such manner

¹ Delafond was the first who tried the cultivation of anthrax rods. By exposing virulent blood to air he saw that these little sticks became lengthened, and he recognized that a true cryptogamic vegetation was taking place; he attempted "to obtain a complete development of these productions in order to make them produce spores or granulations;" but, he added, "notwithstanding the various and numerous experiments made, I have not as yet been able to attain this important result." (See Bull. Soc. cent. vét., 1860, p. 726.)—N. D. T.

that we may observe a free ovaliform space between two joints, when they are united end to end. A series of segmentations arranged in such manner form a linear figure which has the aspect of bamboo. Bacteridia are found in all the tissues of cadavers affected by anthrax, especially in the viscera. Their reproduction occurs by transversal segmentation and by growth in their length (asporogenous bacilli).

Outside of the organism the bacteridia are transformed into intricate filaments; they never become ramified and may acquire one hundred times the length of the primary element. At determined intervals we see the appearance of ovoid corpuscles which are very refringent—the SPORES; there the protoplasma of the filaments becomes granular, the covering membrane is destroyed and the spores are freed (sporogenous bacilli).

The bacteridium may be cultivated in various mediums (gelatin, bouillons, potato, agar, etc.). The cultures upon a surface of gelatin show a flaky, grayish-white aspect; they liquefy this substance; when examined with a low-power magnifying glass they appear composed of an irregular packing of filaments which extend beyond the borders of the colony, afterward become twisted and re-enter the mass; the arrangement of these filaments resembles the twists of a whip-cord. The cultures made by puncture form, on each side of the track made with the needle, a whitish-gray band with fine ramifications, which seem to be formed by small crystalline agglomerations; gelatin also becomes liquefied. Cultures upon potatoes appear under the aspect of a whitish-gray lining; the bacilli in it are provided with ovoid spores. Upon agar the cultures have a bluish-white color, with dull reflex. In liquid mediums (serum) we obtain grayish flaky masses which cover the bottom of the bottle.

The bacteridia take the basic colors of aniline quite easily, but especially the alkaline solution of methylene-blue. When only the spores are to be colored we must first submit the preparation for fifteen seconds to the action of concentrated sulphuric acid and afterward wash it (Buchner).

THE BIOLOGICAL PROPERTIES OF THE BACTERIDIUM are not less interesting than its morphological characters. We must particularly indicate the influence which is exerted upon it by the culture-medium, oxygen, temperature, and certain antiseptic agents.

a. The mediums in which the bacteridium may vegetate are:

the blood, the plasma of the blood and the serum, the transudates, the aqueous humor, milk, and various other secretions and excretions of the mammals, animal matter and excretions mixed with the soil (especially those of the ox), decoctions of meat, neutral or slightly alkaline, gelatin, peptonized gelatin, raw or cooked potatoes, slightly alkaline decoctions of hay and those of a large number of grains mixed with chalk (in order to neutralize the acids), the infusion of peas; the juice of carrots, turnips, potatoes; crushed grain, particularly wheat, and leguminous plants. In order to develop it also requires a determined quantity of water. When the proportion of this exceeds 1 to 20 vegetation is retarded. In distilled water and in drains, at the temperature of the cellar, it dies within a day (Hochstetter); it loses its virulence rapidly at the temperature of the room or of the oven (Meade Bolton); it perishes rapidly in water containing a certain proportion of organic substances (in water of the Panke, for instance), and at the temperature of spring-water (Wolffhügel and Riedel). It does not resist desiccation. In the very thin layers of the organic tissues and in blood-clots desiccation destroys its vitality in from two to three weeks; when the blood forms a very thick layer it loses its virulence only in from four to five weeks.

b. The bacteridium being *aërobic*, the oxygen of the air is absolutely indispensable to its development. In cadavers affected by anthrax sporulation does not take place, because oxygen is wanting (Koch). Inside of the meat it forms neither spores nor filaments, even when during the summer the meat is exposed for several days to a high temperature (Johne).¹ If preserved in hermetically-sealed tubes, blood of anthrax loses its virulence within eight days.

c. The temperature plays a very important rôle in the development of the bacteridium. The height of vegetation is observed at a temperature of 35° C. Growth stops below 12° C. and above 45° C. In the soil at a certain depth this phenomenon is produced because the temperature is always lower than 12° C. The bacteridium loses its virulence only at 55° C. Pasteur has ob-

¹ Johnne has claimed that anthrax meat does not contain spores, and that the infection produced in man by consumption of this meat, which is always caused by the bacteridium, could only take place by the mouth and pharynx. But Schmidt-Mühlheim has shown that if sporulation does not take place in the meat it may happen upon its surface.—N. D. T.

tained his virus by cultivating it for some time in an atmosphere of 42° to 43° C., and in the presence of oxygen. Toussaint has produced his by subjecting it in defibrinated blood at a temperature of 50° to 55° C. It is very rare that the virulence of anthrax is destroyed by cold. According to Feser, the cold of winter has no influence upon it if the temperature is not lower than 10° C. for at least three consecutive days. Gibier has obtained a mitigated virus by subjecting the cultures to the action of a temperature of 45° C.

d. Light (especially sunlight) hinders the development of the bacteridium (Arloing).

e. Putrefaction kills it within a certain time; the normal gastric juice destroys it also.

f. Among the chemical agents which hinder its vegetation we must mention sublimate (1 : 300,000 to 1,000,000), essence of mustard (1 : 33,000), arsenite of potash (1 : 10,000), iodine (1 : 5000), bromine (1 : 1500), salicylic acid (1 : 1500), phenic acid (1 : 1000), boric acid (1 : 800), quinine (1 : 600), etc. Sublimate (at 1 : 30,000), cresol (1 : 15,000), phenic acid (1 : 200 to 100), thymol and salicylic acid (3 : 1000), permanganate of potash (1 : 1000), sulphuric acid, alcohol, etc., destroy its virulence entirely. Iodoform does not seem to have any influence upon it, neither in the organisms nor in the various mediums where it exists.

2. The reproduction of the bacteridium outside of the organism is performed by lengthening of the bacilli, by granular transformation of their protoplasm, and the appearance in this of small corpuscles which are very refringent and soon constitute true ovoid *spores*. The spore in its turn gives birth to the bacteridium by a kind of sprouting; upon one of its poles we see the appearance of a prolongation, at first oviform, then cylindroid, which becomes elongated into a bacillus, while the spore itself becomes gradually smaller, and then disappears completely.¹ When the spores reach the soil they may preserve their virulence and their power of reproduction for more than six months. The bacteridium may also vegetate in the soil and produce spores. The extreme resistance of these to destructive influences explains the existence of regions which are constantly infected and designated under the name of

¹ In order to produce vegetation of these spores and their transformation into bacilli, it is indispensable that they be transferred to a new medium of culture.—
N. D. T.

anthrax stations (damned fields). The spores remain intact for many years, and may be transported to great distances in food, soil, water, etc.

The bacteridium is thus at the same time *endogenous* and *exogenous*, whence anthrax is a miasmatic, infectious, and miasmatico-contagious disease.

THE BIOLOGICAL PROPERTIES OF SPORES are essentially different from those of bacteridia. The formation of spores requires oxygen at a certain temperature; the most favorable is 35°C . Sporulation does not take place below $+12^{\circ}\text{C}$. nor above 43°C .; it is hindered by light, especially sunlight. On the other hand, the spores are little sensitive to the action of water; they are resistant to the temperature of boiling water, intense cold, and for years to desiccation. A temperature of $+110^{\circ}\text{C}$. when prolonged for ten minutes, and a cold of -110°C . lasting for hours, have no influence at all upon them. Among the disinfecting agents which attenuate or destroy their vitality we may especially mention sublimate, cresol, chlorine, bromine, and iodine. They are killed within ten minutes by a solution of sublimate at 1 per 1000; within twenty-four hours by a 2 per cent. solution of chlorine, bromine, or iodine; within forty-eight hours by a 3 per cent. solution of cresol (Eisenberg).¹

General pathology. In a majority of cases anthrax is of "miasmatic origin;" in some others contamination is determined either by the secretions or excretions of a sick animal, or by the intermediation of man, insects, or soiled utensils; it is exceptional to see it take place directly from a diseased to a healthy animal.

The bacteridium may penetrate into the organism in three ways: by the gastro-intestinal canal, the lungs, the skin and the integument of natural orifices. In the ox infection takes place, as a rule, by the digestive tract; in the horse and the sheep it occurs sometimes through the digestive apparatus and also by the skin.

1. *Intestinal infection* generates the forms of anthrax designated by the expressions *intestinal anthrax*, *alimentary anthrax*, *spon-*

¹ Lignières has studied the action exerted on anthrax spores by the principal antiseptics. Silkworm-gut impregnated with these spores and dried has been dipped in solutions, then withdrawn, washed and placed in pure bouillon. The results obtained were:

The spores germinated after an immersion of twenty days in cresol at 5 per cent.; after twelve days in phenic acid at 5 per cent., and after nineteen days in lysol at 10 per cent. They have remained sterile after an immersion of forty-eight hours in a 1 to 15 solution of solutol, and of a few minutes in sublimate at 1 per 1000.—N. D. T.

taneous or *internal anthrax*, *anthrax without any external affection*, *anthrax without localization*, *anthrax fever*, *anthrax apoplexia*, etc. As we have stated, it is common in the ox. It is usually produced by spores which are ingested with food or drink. This mechanism of infection is particularly frequent during the course of epizootics. The virulent elements penetrate the mucous membrane of the intestinal grêle,¹ even when it is intact, and thus enter the circulation. The spores resist the acid gastric juice, which is fatal to the bacteridium. They are generally introduced into the organism by the intermediation of fodder coming from regions which have been infected for a long time, or at a recent date, by the burying of anthrax cadavers or remains. In some instances the spores contained in the superficial layer of the soil are spread upon plants by rain; at other times they are deposited there by dust; they may exist in ground adherent to roots, turnips,² etc., in well water, swamps, pools, ponds, in those which are close to tanneries or establishments where wool is washed. Carnivorous animals become easily infected by eating cadaveric remains, and sucklings through the mother's milk. Infection by the spores may also take place when the pavement of the stables is renewed, or even by the litter and by virulent earth. Pasteur has recognized that the spores of anthrax are brought to the surface of the ground by lumbricoids. Koch has contested this fact; according to him, the temperature of the soil at a depth of 0.50 millimetre to 1 metre would be too low to allow the formation of spores; experiments have demonstrated to him that the intestinal contents of lumbricoids which were kept for some time in centres of anthrax are not virulent. But Bollinger has experimentally confirmed Pasteur's theory; 5 per cent. of the worms coming from an infected pasture contained spores of anthrax.³

2. *Cutaneous infection* produces cases of anthrax which are designated under the names *inoculating anthrax*, *external anthrax*, *anthrax with localizations*, *carbuncular disease*, etc. This form of

¹ In experiments which Pasteur and Toussaint made in Beance (1878) they recognized that inoculation of anthrax frequently takes place by the buccal and pharyngeal mucous membranes.—N. D. T.

² In 1887, in a farm of Eure-et-Loire, out of eleven bovine animals ten died of anthrax which had been contracted by eating turnips coming from a piece of ground in which it was the custom to bury cadavers of sheep having died from anthrax.—N. D. T.

³ Other lower animals play a rôle in the propagation of anthrax similar to that of the lumbricoids. Karlinski, in his experiments made with cellar and field snails, has recognized that the intestine of these animals also contains living spores eleven days after ingestion of the food which contained them. (Roger: *Treatise of Medicine*.)—N. D. T.

contamination, which is much more rare than the preceding, is generally only observed in sporadic anthrax. The bacteridium or spores penetrate into the organism through wounds of the skin or the integument of the natural orifices. Infection may take place in pastures, by the intermediation of utensils or instruments soiled by virulent matter, by bites (*shepherd dogs*), in performing operations (bleeding), by furs or skins (even white leather), by insects (*Musca domestica* and *M. vomitoria*), by certain varieties of *Tabanus* and *Simulium*. Bollinger and Zeillinger have communicated anthrax to rabbits by inoculating them with the matter obtained by crushing flies taken from a cadaver affected with anthrax.

3. *Anthrax produced by inhalation* is the rarest form. In this mode of infection the spores are brought upon the pulmonary mucous membrane. Experiments have demonstrated that the healthy respiratory apparatus may become infected in this manner (Feser, Buchner, Lemke). Cadeac and Mallet have obtained only negative results.¹

In cutaneous anthrax the spores are transformed into bacilli in the same location (skin and subjacent connective tissue); then generalization takes place through the circulation.² Their pullulation is especially active in the blood. Rodet has recognized the presence of bacilli in the blood in less than one hour after inoculation. The bacteridia exist in very large numbers in the capillary plexus of internal organs (spleen, intestinal mucous membrane, mesentery, mediastinum, lungs), but it is often impossible to find these in the blood of the large peripheric vessels. By accumulating in large numbers in the capillaries they may produce lacerations of these canals and hemorrhages. The bacterian emboli generate in the internal organs as well as in the skin, carbuncular tumors, and cedema, accompanied by gelatiniform extravasations and abundant leucocytic diapedesis.³

The anthrax character of the disease known as *wool-sorters' disease*, and some of the cases of the disease of rag-pickers (*Hadernkrankheit*), have clearly established that the mode of entrance of the virus is through the respiratory organs.—N. D. T.

² The experiments of Cohn have demonstrated that the inoculated bacteria at first invade the lymphatic system; they are arrested in the various ganglions upon their course, and are finally poured into the blood; in the ganglions which they traverse they produce certain constant lesions: hyperemia, tumefaction, hemorrhages, and cedematous infiltration, which permit us to recognize their mode of introduction into the organism.—N. D. T.

³ Chambrelent and Moussons have recognized that the bacteridium may pass into the milk of females affected by anthrax (*Comptes rendus de l'Academie des sciences et Recueil Vét.*, 1884).—N. D. T.

THE ACTION OF BACTERIDIA IN THE BLOOD is as yet imperfectly known. Besides the theory of absorption of oxygen, Bollinger has adopted that of stasis in the capillaries; it explains the hemorrhages, but leaves in obscurity the pathology of other symptoms. They have at all times opposed the mechanical to the chemical theory, according to which the products secreted by the bacteridium cause the symptoms of anthrax. A series of objections have been made to the latter. Klebs, Pasteur, Nencki, by filtering cultures of anthrax through porcelain under pressure, obtained a non-virulent liquid. The inoculation of blood, of the substance of the liver, of the spleen, etc., coming from the foetus of females which were affected by anthrax has never produced anthrax; thus in the tissues and putrid fluids of the foetus the bacteridium is not found, but only the noxious principles dissolved in the maternal blood.¹ More recently Hoffa succeeded in isolating the products produced by the bacteridium; these basic products appeared very toxic; when injected in animals they induced symptoms of anthrax. He calls these "alkaloids of anthrax," and considers them as similar to neurine of the ptomains of Brieger.

GENERALITIES UPON THE FREQUENCY OF ANTHRAX. Anthrax is most commonly observed in the ox. Carnivorous animals are rarely affected by it. If considered in relation to their receptivity for anthrax virus, the animal species are classified in the following order: ox, sheep, goat, horse, stag, antelope, deer, and camel; then come the cat, rabbit, guinea-pig, mouse,² hare, tame rabbit; finally, the dog, pig, and fox—these animals are only slightly susceptible (most observations reported under the name of anthrax of the pig are related to swine plague). The rat seems to possess almost complete immunity from inoculation and ingestion of virulent matter. The duck, chicken, and pigeon contract anthrax(?), while birds of prey are refractory to it. Fish and amphibia are hardly affected by it. Well-fed animals are predisposed to it,

¹ A possibility of transmission of anthrax from the mother to the foetus, recognized by Strauss and Chamberland in 1882, has been since confirmed by numerous authors. According to Malvoz, this transmission is produced only when placental alterations exist. By inoculating anthrax in the unborn fetus of the rabbit Lingard has obtained variable results; sometimes the mothers were not infected and became refractory; in other cases they died from anthrax; in the latter case we have always found placental alterations (Roger, loc. cit.).—N. D. T.

² Guinea-pigs and mice are extremely sensitive to anthrax. According to W. Chene, a single bacteridium deposited in the connective tissue suffices to kill this animal.—N. D. T.

especially at the beginning of epizootics. Newly-imported exotic animals are more susceptible than acclimated subjects.¹ A first infection confers partial immunity.

Anthrax is sometimes observed in a sporadic state, and also as an enzootic or epizootic. It usually appears as an infectious enzootic disease, which is stationary in some districts. Its development depends upon a certain composition of the soil and determined conditions of moisture, temperature, and vegetation. Anthrax is a miasmatic infection, and preferably exists in countries with dark, light soil, rich in organic matter; in regions with clay, lime, or calcareous soil which contain a small proportion of organic remains; in those with a swampy, turfy character, and where the subsoil is impermeable. Enriching of the soil with mineral substances (lime, plaster), or organic matter (manure, deposits, mud coming from ponds), favor the development of bacilli (Nocard).² The appearance of anthrax is also influenced by the degree of moisture of the soil. Certain modifications which occur in the condition of this are very favorable to it. It is sometimes observed in wet, swampy grounds when they become partially dry during the hot season. Wald, Buhl, Reinelt, Friedrich, and Soyka have observed that the cases of anthrax are so much more numerous in proportion to the low level of underground waters and scarcity of rain. The same law applies to soils rich in mould; these are the most favorable for the development of bacteridia when an elevated atmospheric temperature, after heavy rains or inundations, determines a very rapid decrease of the level of underground waters. This is the reason anthrax is so frequent in low lands exposed to submersion. The conditions of temperature are also very important. The biological

¹ Anthrax infection is favored by numerous circumstances, but particularly by young age and various causes of weakening of the organism. Strauss has recognized that the animals are much more susceptible to anthrax in proportion to their young age, and that the receptivity is greater in a very young pup than in an old guinea-pig. Charrin and Roger have demonstrated that extreme exertion lessens considerably the resistance to infection: in 13 white rats inoculated and subjected to fatiguing exercise 11 died from anthrax; of 8 check animals which were left at rest after inoculation 6 resisted.—N. D. T.

² I have been able to obtain a very remarkable case of infection of the soil by cadavers which were buried in a piece of woods. The ground, which was cleared more than ten years after and turned into a meadow, had remained infected. I have observed, on the other hand, two examples of infection of the soil by an artificial substance containing blood. Two magnificent flocks of sheep were decimated by anthrax after the use of that manure: in one case in a natural meadow, in the other upon a soil which was under cultivation with lucern.—L. T.

properties of the bacteridium permit us to understand why anthrax makes most ravages in the summer, from June to September, while it does not exist in winter.

Anthrax infection is found in the five divisions of the world and in nearly every country. The cartographic tableau established by the German sanitary service for the years 1886 and 1887 shows that it existed mainly :

1. In several districts in the neighborhood of the River Pregel which centres around the district of Gerdanen.

2. In the lower Vistula (Thorn and Mohrungen).

3. In the regions of the Netze, Warthe, and Oder (Wohlan-Trebnitz, Pyritz).

4. In the districts of the Elba and Saale, in Saxony, Thuringia, in the mountains of Lansitz, the forests of Thuringia and the Harz (Sangerhausen, both districts of Mansfeld; also Zwickau and Lobau).

5. In the regions located between the Mein, the upper Danube, and the Rhine, in the Odenwald, the Black Forest, the Alps, Suabia, and upon the highlands of Franconia (Uffenheim, Marbach, Heidelberg).

6. Between the Mein and the Sieg, the Rhine and upper Fulda, in the Vogel Mountains, Rothnaar and the dependencies of Westerwald (Friedberg as central point).

7. In the high Ems district (Wiedenbrück).

8. In the mountains of the Vosges and Haardt, the plains of the Rhine, Alsace-Lorraine, the Palatinat and Eifel Mountains (principal centres : Sarregue mines, Saberne, Enskirchen).

9. In the highlands of Suabia and Bavaria, between the high Danube and the River Lech, as well as all the districts situated south of the Tsar and Inn Rivers.

In Prussia anthrax is particularly observed in the province of Saxony and in Thuringen. The districts of Hettstädt and Mansfeld are those where it makes most ravages. This region, which is inundated every year, is formed in part by a calcareous soil which is porous, black, rich in mould, and which has a gravelly subsoil, and by swamps. During the dry season the animals have frequently nothing but rain-water, which is stagnant and putrid, for quenching their thirst. Anthrax exists permanently in the region of Merseburg, the valleys of the Saale, Elster, Mulde (regions where the good soil is one foot thick and which are inundated

every year), in the province of Brandenburg, in the neighborhood of Potsdam (swampy flat lands, sandy, turfy lands rich in mould), in Hessen-Nassau, and in the valleys of the Rhine (Cleve, Cologne, Bonn), of the Röhr, of the Erfte (turfy swamps), of the Sarre, in the Eifel, in the province of Posen (diluvian soils with impermeable subsoil), and in Silesia.

In Bavaria its principal centres are Miesbach, Töls, Werdenfels (these localities are situated at an altitude of 1100 to 1300 metres). In these Bavarian Alps there are numerous swamps; we find everywhere a thick layer of mould mixed with calcareous substances. The neighborhood of human settlements contains much organic matter and spring-water at a temperature of 10° to 12° C. Anthrax is frequent upon the banks of the Danube, where vast regions are often submerged and covered with reeds and moss. It is also found in the valleys of the Tsar, Lech, and Tun.

In France anthrax is particularly common in Beauce (Eure and Loire, Loiret), in the Department of Seine and Marne, in Sologne, Burgundy, Poitou, Guyenne, Languedoc, Forez, Lyonnais, Auvergne, Dauphiné, and Provence.

In Russia it is frequent in Siberia, in the governments of Astrakan, Orembourg, Perm, Novgorod, St. Petersburg, Archangel, Finland, and Lapland. It exists also in Switzerland, Austria, Italy, England, Spain, Turkey, Africa, America, in the East Indies, in Persia, China, and in many other countries.¹

In some years the losses occasioned by anthrax are considerable. In northern Bavaria, from 1872 to 1875, they amounted to \$75,000 (900 oxen and 40 horses). In 1874, in the district of Potsdam, anthrax killed 2000 wild ruminants. In Prussia, from 1878 to 1883, it caused damages estimated at \$75,000 (more than 6000 oxen, 3000 sheep, 300 horses, without counting the numerous cases not included in the official reports).

In France, where it claims fewer victims than formerly, its inroads amount to millions yearly. About 1830 the annual losses in Beauce were estimated by Delafond at \$2,000,000 (nearly 5000 oxen and 300,000 sheep). In the Department of Seine and Marne since 1859 the yearly loss is \$100,000, and in the Department of Aisne nearly \$24,000. In the government of Pskow, in 1884, it

¹ Loir, Germond, and Hinds have recognized that the epizootic disease designated for a long time in Australia as *Cumberland disease*, and which caused losses amounting to 300,000 sheep yearly, is the bacteridian anthrax.—N. D. T.

killed 4000 horses, nearly 2000 oxen, and 1000 head of small cattle (W. Koch). In that of Novgorod, in 1867-68, it killed 40,000 horses, more than 800 cows, 6000 sheep, and 500 human beings (Grimm).

General anatomical alterations. The principal alterations produced by infection of anthrax are :

1. Hemorrhages in nearly all the organs ;¹
2. Sero-gelatinous and hemorrhagic infiltrations of the subserous connective tissue (mesentery, mediastinum), of the submucous connective tissue (intestine), and of the subcutaneous connective tissue ;
3. Tumefaction of the spleen and parenchymatous inflammation of the principal viscera (liver and kidney) ;
4. Muddy consistency of the blood, poikilocytosis, and leucocytosis ;
5. The presence of bacteridium in all the tissues, but especially in the blood of the principal viscera (spleen, intestines, liver) and in the mesenteric infiltration.

The various organs present the following lesions :

The cutaneous vessels are obstructed with black, muddy blood ; a minute examination of the derma shows small hemorrhagic centres which may elevate the epiderma and form vesicular embossments. In cases of anthrax consecutive to experimental or accidental inoculation we see on the skin hard nodes, which vary in size from a lentil to a kidney bean, and the tissues of which are partially necrosed (anthrax tumors). At the seat of this anthrax oedema the integument often becomes gangrenous over large surfaces.

The subcutaneous connective tissue shows more or less extensive and abundant hemorrhages. In cases of oedema of anthrax we find therein circumscribed or diffuse suffusions of somewhat firm consistency and variable dimensions ; their color may be orange-yellow or brownish-yellow and studded with reddish spots ; we may also find bloody purulent infiltrations. The neighboring lymphatic ganglions are hypertrophied, oedematous, or marked by hemorrhagic centres. The infiltrations are particularly abundant in the subcutaneous connective tissue of the neck and around the trachea.

The muscles have a yellowish, brown-red, dark-red, or violet shade ; they are overrun with small hemorrhages ; their tissue is

¹ Reynal has pointed out that these interstitial hemorrhages are inconstant, and, in fact, they are mostly wanting in somewhat firm tissues. When they are absent we must not conclude that anthrax does not exist.—L. T.

very friable. The myocardium presents similar alterations (parenchymatous myocarditis).

The splanchnic cavities (peritoneum, pleura, pericardium) contain a small quantity of bloody transudate. The subserous connective tissue is infiltrated, especially in the neighborhood of the kidneys. In the mediastinum, the mesentery, and under the epicardium we find bloody extravasations of variable dimensions. The lymphatic ganglions (mesenteric and mediastinal) are greatly tumefied, infiltrated, and ecchymosed. The internal organs (liver, spleen, kidneys, and lungs) are hyperemic. The large veins and the heart are filled with black blood; the endocardium and vascular endothelium are markedly red and impregnated with coloring-matter which is dissolved in serum.

The spleen is sometimes uniformly hypertrophied, at other times deformed by prominent tumors; its loosened capsule is much distended by extravasated blood; at times we find therein small vesicular embossments with bloody contents; its pulp is softened, liquid, and dark-red.

The liver and kidneys are hyperemic, tumefied; their parenchyma is overrun with hemorrhages, their regular cells are affected by various degenerative processes (parenchymatous hepatitis and nephritis).

The lymphatic vessels of the portal system are distended and the retro-peritoneal (perirenal) tissues are tumefied and dropsical. The peritoneum is inflamed; the subperitoneal tissue (intestines, abdominal walls) is infiltrated with blood and serum.

The intestinal canal shows lesions which vary according to the way the infectious agents are introduced.

In *cutaneous anthrax* the intestine is often absolutely normal; sometimes, however, we observe submucous and subserous hemorrhages and obstruction of the mesenteric ganglions.

The principal lesions of *intestinal anthrax* exist in the duodenum; the large intestine is more rarely affected. In the benign forms of this latter variety the mucous membrane is the seat of a circumscribed or diffuse obstruction; it is spotted red and overrun with hemorrhages, covered with erosions upon the surface of Peyer's patches and also the solitary follicles; the bacteridia are sometimes found in considerable quantities upon its surface; in some points where they are extremely numerous this membrane is gangrenous and ulcerated. In very serious forms the stomachal mucous membrane

shows bloody infiltrations; the membrane of the abomasum and duodenum is dark red and covered with erosions, ulcerations, or affected by necrosis; it frequently forms thick, quivering swellings (œdema of anthrax) when the submucous connective tissue is infiltrated with bloody serum. On the surface of Peyer's patches and solitary follicles we observe flattened or irregular tumors, which are rich in bacteridia and the surface of which has the aspect of diphtheric lesions. The intestinal contents are bloody. The mesenteric ganglions are obstructed and infiltrated with serum and blood. We observe more rarely similar alterations in the rectum, where they are ordinarily developed consecutively to wounds made with the nails; the mucous membrane is disposed in narrow folds and partially necrosed; its surface is bloody.

In the respiratory apparatus the lungs are congested, ecchymosed, and œdematous. The entire mucous membrane is red and strewn with ecchymotic spots; that of the larynx and the origin of the œsophagus is infiltrated (angina of anthrax); sometimes the opening of the laryngeal duct is considerably contracted; the trachea and bronchi contain bloody mucus.

The brain is hyperemic and overrun by hemorrhagic centres or ecchymotic points. Sometimes there are extensive hemorrhages in the meninges and a bloody serum in the ventricles. In the anterior chamber of the eye and under the retina we may find bloody extravasations.

The genito-urinary organs, the salivary glands, the thyroids, the bones, the articulations, etc., are marked by hemorrhagic lesions. The urine is frequently bloody.

The blood is uncoagulated, muddy, dark red, as if varnished upon its surface; when in contact with the air it does not assume its vivid red shade. At the microscopical examination the red corpuscles appear in irregular forms, extremely varied, corresponding with more or less advanced phases of their destruction (poikilocytosis); the number of white corpuscles is much increased (leucocytosis). Between the corpuscles we find bacteridia.

The cadavers become cold very slowly, and are soon much swollen; rigidity of the cadaver is wanting; decomposition follows rapidly. The mucous membranes have a red-blue coloration; the natural orifices (mouth, nose, and anus) give exit to blood or to bloody mucus. Rectal prolapsus is frequent.

These various alterations may be wanting in cases with subacute

course (apoplectiform anthrax). But a microscopical examination of the blood always reveals the presence of bacteridia.¹

General symptoms. The manifestations of infection by anthrax vary with the species, the individuals, and according as to whether the process is localized in the intestines, the skin, or the lungs. Intestinal anthrax itself may be marked by variable clinical manifestations, and cutaneous anthrax is sometimes accompanied by metastatic alterations of the intestines, the serous membranes, etc. The quantity of virus introduced into the organism also exerts a considerable influence upon the symptomatic tableau. The disease may finally have an abortive or remittent course.

No matter what the form of infection may be, the invasion is always sudden, abrupt, and the evolution precipitant and tumultuous. The general condition is greatly affected and the fever very intense; we frequently observe hemorrhages upon the mucous membranes, also tumefactions, œdema of the skin, intestinal and cerebral troubles, and dyspnoea. An examination of the blood or of the serum of the œdema may permit us to recognize the bacteridia. Death occurs, as a rule, within one to three days. Observers have established in anthrax divisions based upon the course of the disease (*Anthrax acutissimus*, *acutus*, and *subacutus*), or upon the presence or want of localizations. This latter grouping seems to us preferable from a clinical standpoint.

A. FORMS OF ANTHRAX WITHOUT ANY VISIBLE LOCALIZATIONS. These are generally the result of an infection produced by the spores. By making animals ingest these they may be produced experimentally. According to their course, they are distinguished as *hyperacute*, *acute*, and *subacute*.

1. The *hyperacute forms* are known under the names of *anthrax apoplexia*, *apoplectic* or *fulminating anthrax*, *disease of the blood*, *anthrax acutissimus*. Their symptomatology is that of cerebral apoplexy. The animals are affected suddenly, stagger, and fall; the nose, anus, and mouth give exit to bloody liquids. The patients die in convulsions within a certain lapse of time, which varies from a few minutes to one hour at most. They are often found dead in the morning on opening the stable; they sometimes die during work, on pasture, or while taking their meals. This variety

¹ The bacteridia are less numerous in the blood in proportion to the more rapid course of the infection. In cases with subacute evolution often the spleen is not hypertrophied.—N. D. T.

of the infection is most commonly observed in the sheep and the ox, especially in the beginning of epizootics.

2. The *acute forms* evolve a little less rapidly. Their duration is generally from two to twelve hours—twenty-four hours at a maximum. The fever becomes rapidly intense (40° to 42° C.). Sometimes we observe symptoms of cerebral hyperemia: anxiety, excitement, stamping, bellowing, rabiform phenomena, convulsions, spasms, grinding of teeth, stupefaction, weakness, staggering gait; finally, *apoplectiform*, death; in other cases those of pulmonary congestion: accelerated and laborious breathing, groans, complaints, cardiac palpitations; a small accelerated pulse, which is almost imperceptible; cyanosis of the mucous membranes of the head, bloody emissions by the natural orifices; also hematuria petechiæ of the conjunctiva,¹ convulsions, staggering gait, and death by asphyxia. Sometimes these tumultuous manifestations become attenuated or even disappear partially, but return soon after (remittent anthrax). In some cases they are announced by certain prodromes: depression, slight digestive troubles, constipation, tenesmus, etc.

3. The *subacute forms*, which are described under the names of *anthrax fever*, *intermittent anthrax*, etc., are the most common in horses and cattle. Their clinical characters, which are nearly similar to those of acute forms, are, however, more distinct, better defined, and their course is less rapid (twenty-four to forty-eight hours on an average; five to seven days on the maximum). The febrile symptoms are more marked (chills, irregular distribution of the temperature, general troubles); the same is the case with phenomena due to pulmonary or cerebral congestion. Serious intestinal symptoms are often added to these manifestations (colics).

Remissions are even more frequent than in the preceding forms.

B. FORMS OF ANTHRAX WITH VISIBLE LOCALIZATIONS. They generally result from bacillary invasion. As in the others, they may be produced experimentally.

Cutaneous anthrax tumors and *œdema* have been for a long time designated by the expressions of *carbuncular disease*, *carbuncular fever*, *black variola*. They are especially observed in the horse and the ox; they exist also in the dog. Cutaneous tumors are circum-

¹ Reynal has denied the existence of these petechiæ, and, in fact, they do not ordinarily exist. They are mentioned in old works, perhaps because they have been confounded with true anthrax anasarca, also called white anthrax.—L. 7.

scribed, hard, hot, and painful in the beginning; later, they become gangrenous, cold, and insensitive to pain. Anthrax œdemas are diffuse, doughy, fluctuating, cold, and indolent cutaneous tumefactions. The duration of the disease varies from three to seven days. The cases of recovery are more numerous than in the other forms (?). Fever may appear before or after the development of tumefactions.¹

Anthrax tumors and œdema of the mucous membranes are observed in the mouth (gloss-anthrax), in the larynx and pharynx (anthrax of angina), in the rectum (*sang de lombes*). They are accompanied by a more or less intense fever, and, according to their location, they determine various troubles: dyspnœa, laryngeal contraction, dysphagia, general cyanosis, tumefaction of the submaxillary, neck, chest, and violent expulsive efforts, etc. Death occurs within from twelve to twenty-four hours. This is the form usually taken by the infection in the pig and the dog.

In the ox we find most frequently anthrax fever, anthrax apoplexia, and anthrax tumors; the horse shows the same forms as the ox, and more particularly the first; in the sheep we observe especially anthrax apoplexia; in the dog, anthrax tumors; in the pig, anthrax angina and gloss-anthrax. Most cases of anthrax of the pig cannot be accepted without reserve.

Diagnosis. In the acute and subacute forms the *intra-vitam* diagnosis is based upon the finding of the bacteridium and on the infectious character of the disease. Quite often the bacteriological examination of the blood gives a negative result because the bacteridium usually locates in the internal organs; it has therefore been advised to obtain the blood from the liver by means of an exploratory puncture made with the trocar. During life the diagnosis is at times very difficult. In some cases the symptoms of anthrax have a somewhat great similarity with those of pulmonary or simple cerebral congestion,² poisoning, septicemia with abrupt appearance, etc. The disease is generally only recognized after death. The macroscopic observations are almost always insufficient. The anatomical

¹ Besides the lesions which occur in the neighborhood of the point of inoculation, the tumors and anthrax œdema are generally the result of accidental contusions having determined a loss of blood in the subcutaneous tissues. The absence of crepitation in these tumors permits us to distinguish them from those of symptomatic anthrax.

—N. D. T.

² In such cases the rising of temperature distinctly differentiates anthrax from simple congestion.—L. T.

diagnosis absolutely requires a search for the bacteridium. It may be made with or without previous staining. The technique of this is very simple; we spread a drop of blood upon a cover-glass and dry the preparation; then we deposit two or three drops of a solution of methylene-blue or gentian-violet; after a few minutes we wash with water and mount it in Canada balsam.

Inoculation is another means of diagnosis. As agents we may use mice, rabbits, guinea-pigs, or sheep. Death occurs within from two to three days, and it is easy to find the bacteridium in cadavers. This process enables us to recognize "intestinal anthrax" during life. It is sufficient to introduce into the cutaneous derma a particle of suspected matter (blood, mucus, excrements, etc.). We must avoid the displacement of the virus by the blood which escapes from the wound. Inoculation is not successful when using non-virulent blood coming from peripheric vessels or even from large vascular bodies; it also gives negative results when the matter containing the bacteridium is soiled by other microbes or when it is in a state of putrefaction. All species are not equally adapted for testing these inoculations, and some animals seem to possess individual immunity from anthrax. It ought to be a rule to inoculate simultaneously several subjects of different species.¹

Differential diagnosis. We will only deal here with bacilli which show a certain morphological analogy with the bacteridium. For a differential diagnosis between anthrax and the diseases which may simulate it, see the chapters which comprise the study of this infection in the various animal species.

1. The bacilli of septicemia and putrefaction (among these latter the *Bacterium termo* especially) are generally animated by true movements, and their extremities are rounded. There are some,

¹ In doubtful cases the diagnosis may be established by bacteriological examination, inoculation, or culture. The bacteridium when immobile easily takes Gram's or Weigert's test. We may say that the guinea-pig always dies from inoculation of bacterian anthrax. Often the bacteridium appears in the blood but a few hours before death; this is a fact which is already pointed out in Delafond's works. In order to decide with certainty the virulence of this liquid we must resort to culture, for when the bacilli are not numerous in it they may escape a microscopic examination. By placing a culture of one drop of pure blood in bouillon the bacteridium is developed by forming characteristic flakes. The *bacterium Chauvvi* and the septic vibriones are anaërobic and do not grow. If they are inoculated by means of a puncture in gelatin, the bacterium vegetates along the track made by the needle, and especially on the surface of the layer of gelatin, which becomes liquefied. If planted on potatoes, it soon forms a grayish-white layer having a peculiar velvety aspect. (See Symptomatic Anthrax.)—N. D. T.

however, which much resemble the bacteridium. In doubtful cases we must resort to the processes of staining and try to establish the characteristic disposition of the extremities of the anthrax bacillus, or make test inoculations (Kitt).

2. The bacillus of symptomatic anthrax is shorter and thicker than the bacteridium; it is rounded at its extremities, and is animated by very active movements; its pullulation is accompanied by escape of gas; in the cultures it forms folded, whitish-gray membranes. It is an anaërobic microbe; it is impossible to cultivate it upon the potato (Kitt).

3. The septic vibrione is thinner than the bacteridium; it does not exist in the blood; its form is rectilinear, not bent, and is animated by slow undulatory movements; it is developed, forming long filaments which are often incurved and non-sporulated. In the cultures it produces gaseous bullæ. If we introduce into gelatin a fragment of tissue containing vibriones, we see around it the development of a small whitish liquid mass, the surface of which seems to be strewn with very fine needles. This micro-organism vegetates with some difficulty; its culture is an operation belonging to the laboratory (Kitt).

4. The hay bacillus (*Bacillus subtilis*) is thick, short, and animated by undulatory movements, and carries upon its extremities a strong flagellum. It is developed upon an ovoid spore and perpendicularly to the longitudinal axis of this. Its biological properties are without importance from a differential diagnostic standpoint; like the bacteridium it is aërobic (Kitt).

Prognosis. Bacteridian anthrax is a very serious disease. Its average mortality is from 70 to 80 per cent. In *apoplectiform* anthrax it is 100 per cent. When the epizootic lasts for a long time its malignant character becomes gradually attenuated. Most animals which have resisted a first infection possess immunity for a variable lapse of time. The cases of spontaneous cure are not very rare.

Treatment. The prophylactic treatment is by far the most important. It is necessary to destroy the cadavers and disinfect the stables carefully. The laws upon contagious diseases indicate the other measures which have to be applied. Incineration of the cadavers, burying at a depth of two metres in a place distant from roads, dwellings, pastures, and destruction of the virus by means of steam, are the best disinfecting methods. The agents enumer-

ated in the sanitary law do not destroy the spores. In order to kill these we must use sublimate (1 per 1000), cresol (3 per cent.), chlorine or bromine water (2 per cent.).

Improvements made in the condition of the soil may contribute in great part to the disappearance of anthrax. It is proper to drain swampy grounds, and favor the exit of the water, also to prevent animals from feeding upon infected pastures or wells. In practice it is often more easy and simple to change the food and drink than to resort to emigration. The medical prophylaxis advises the use internally of hydrochloric acid, phenic acid, and salicylic acid; but these agents are often without effect.

The *curative treatment* comprises a large number of medicinal agents: phenic acid and salicylic acid (10 to 15 grammes for the ox), iodine in the form of Lugol's solution, chlorine water, arsenious acid, phosphorus, hydrochloric acid, ammonia, etc. Davaine has recommended the following preparation: iodine, 30 grammes; iodide of potassium, 60 grammes; distilled water, 360 grammes; for the ox—two tablespoonfuls in one litre of water to be given every two hours. In the subacute forms it is usual to administer alkalis and purgatives, especially calomel in small doses. In cases of pulmonary or cerebral congestion we may practise bleeding.

Anthrax tumors of the skin should be deeply incised and treated with anti-virulent agents applied in the affected tissues. We may also use the cautery.

Emmerich, relying upon the results of his experiments, has advised as prophylactic means, in the beginning of epizootics, to inoculate the animals (oxen, sheep, etc.) with weakened cultures of the micrococcus of erysipelas. This micro-organism stimulates the cells, increases their activity, and allows them to resist the bacteridium much better (hyperactivity of the macrophages and phagocytes of Metchnikoff, which are particularly marked in the spleen). According to Pawlowsky, the *Pneumococci*, the *Staphylococcus aureus*, and the *Bacillus prodigiosus* produce similar effects.

PREVENTIVE INOCULATION. It has been known for a long time that most animals which recover from anthrax possess a more or less complete immunity; we have also recognized that the subjects of certain breeds are refractory to it (Algerian sheep and sheep from Barbary);¹ we have finally observed examples of individual

¹ The immunity of Algerian sheep against blood from the spleen belongs to the breed itself; European sheep brought to Algeria do not acquire it, even after five or

immunity. These facts have given rise to researches which have led to the discovery of means enabling us to create immunity artificially. There is no process which is able to confer this on the guinea-pig, rabbit, rat, and mouse (Löffler). Oemler's experiments have demonstrated that it is very difficult to create it in the horse; in the same subject seven successive inoculations were all followed by specific accidents. Man himself does not become refractory by a first affection. On the other hand, in the sheep and ox it is possible to produce partial immunity, but of a relatively short duration.

The first preventive inoculations were made by Toussaint. This investigator used defibrinated blood of anthrax heated for ten to fifteen minutes at a temperature of 50° to 55° C. He did not secure the true mechanism of immunity which he thought he had thus produced. Pasteur has demonstrated that it is caused by a bacteridium deprived of part of its virulence. Pasteur's virus is obtained by cultivating the bacteridium at a temperature of 42° to 43° C. in presence of oxygen. The preparation of the first virus (the weakest) requires twenty-four days; that of the second, twelve days. They are inoculated at an interval of ten to fourteen days.¹

The tubes containing Pasteur's virus (these tubes are furnished by Boutroux, 28 rue Vaugulin, Paris, at the price of five dollars for 50 cattle or 100 sheep) must be used at one time, and it is important to open them only immediately before the operation. We make the injection by means of a Pravaz syringe, the stem of which is fitted with a sliding gauge, and divided into eight equal parts (it is also furnished by Boutroux; its price is seven dollars). We give here the principal directions concerning the manner of

six generations. It may be overcome by the inoculation of large doses of virus; it is strengthened by successive inoculations. By practising these upon pregnant females during the second period of gestation the lambs may be rendered absolutely refractory to infection of anthrax (Chauveau: *Comptes rendus de l'Acad. des Sciences*, 1880).—N. D. T.

¹ The virulence of anthrax may be attenuated by numerous processes: by the action of heat (Toussaint, Pasteur, Chauveau), compressed oxygen (Chauveau and Wosnessenski), antiseptics (Chamberland and Roux), sunlight (Arloing). We may also attenuate it by cultivating the bacteridium in the blood of inoculated sheep (Metchnikoff) or in the body of the frog (Lubarsch). Immunity against anthrax has been conferred by chemical substances. Chamberland and Roux have been able to inoculate sheep with blood sterilized by heat; they have constantly failed when using the liquid obtained from this sterilized blood by filtration through porcelain. (*Annales de l'Institut Pasteur*, 1888).—N. D. T.

operating. After having shaken the tube which contains the first virus we open it and dip the needle of the syringe directly into the liquid and fill the instrument (previously disinfected). For the sheep we inject the eighth part of the contents of the syringe under the skin of the inner fascia of the right thigh; then we compress the puncture slightly by means of the thumb, in order to prevent the escape of the virus. Within from twelve to fourteen days the second inoculation is made in the same manner with the second virus. For the ox we inject one-fourth of the contents of the syringe. The first inoculation is made behind the right shoulder; the second behind the left shoulder. It is proper to cut the hair previously.¹ It is not advisable to inoculate lambs and calves, or cows which are in an advanced stage of gestation.

Chauveau has invented a process of preparation of virus which is much quicker than that of Pasteur. He heats the anthrax cultures for twenty hours at a temperature of $+43^{\circ}$ C., then for three hours at 47° to 49° C.; he then takes the bacteridia into a new medium, where he cultivates them from five to seven days at a temperature of $+35^{\circ}$ to $+37^{\circ}$ C. (for the development of mycelium and its transformation into spores already attenuated); lastly, in order to complete the attenuation of virulence he heats it at $+80^{\circ}$ C. for one hour. This author has prepared another virus by subjecting the cultures to the action of air or compressed oxygen. Chamberland and Roux have weakened the bacteridium with antiseptic agents; by the action of phenicated water (1 per 600) prolonged for twenty-four days, or by that of bichromate of potassium (1 per 2000 to 5000) prolonged for ten days. Gibier has obtained a virus by bringing the bacteridium to a temperature of -45° C. Kitt has attenuated the virulence by passing the bacteridium through the pigeon's body. Chauveau and Perroncito have replaced the two inoculating fluids, which were necessary till then, by subjecting the bacteridium for five days to a temperature of 37° to 38° C. The method of operating recommended by Chauveau consists of injecting under the skin of the external face of the ear by means of a Pravaz syringe, using one drop of the liquid virus from tubes which have been hermetically sealed.

Pasteur's method of inoculating has been put in practice in almost all the countries of Europe (France, Hungary, Germany, Italy, Holland, Belgium). It has given variable results. We will

¹ See instructions relating to inoculation of anthrax.

simply formulate the conclusions which may be drawn from these inoculations made upon several hundred thousand animals.

1. The preventive inoculation of Pasteur does not agree with sheep. The immunity which it confers is often incomplete, weak, and sometimes void; its maximal duration is about twelve months, so that inoculation has to be repeated every year. The mortality produced by inoculation is relatively high; for the second inoculation it is sometimes 10 to 15 per cent. The lymph furnished by Pasteur is sometimes too active and sometimes too weak, according as the temperature at which the culture vegetated was closer to 42° or 43° C. Koch has advised to control the degree of activity of both forms of virus; the first must be fatal to the mouse, but not to the guinea-pig; the second ought to kill the mouse and the guinea-pig, but not the rabbit. Koch has also recommended to cultivate the virus in solid mediums instead of using liquid substances. Kitt has mentioned a method of this kind which is adapted to the requirements of practice. The weakened or mitigated bacteridia contained in the virus may recover their virulence when the influence of the temperature to which they have been subjected has been insufficient in its effects; it is also possible for them to lose their activity entirely if the virus is kept for too long a time. The various ovine races possess a very irregular sensitiveness to the same virus; for each of these a virus of special activity would be required. Finally, the expenses of inoculation are somewhat high (its cost price for 1000 sheep is nearly \$60).

2. In the ox preventive inoculation is more important from a practical standpoint; it may be recommended for infected regions where anthrax is stationary. The immunity conferred by it is far from being complete in all subjects, and does not last more than a year. But the mortality is much smaller than in the sheep; often it is almost nothing. In general, inoculation is followed by a transient fever and slight general troubles. Let us add, that Pasteur's virus is too weak for the ox; those of Chauveau¹ and Perroncito

¹ Chauveau's virus, which is obtained by compressed oxygen, really gives excellent results. In the inoculation at the Agricultural Institute of Santiago with virus which was sent from Lyons, out of forty-four animals of the bovine species of various ages and origin no accident occurred, either after the preventive or the testing inoculations (Besnard: *Journ de Lyon*. 1885).

The experiments made at Pouilly-le-Fort in 1881 have given proof of the immunity conferred to animals by Pasteur's inoculation, and the results of inoculations made in France and in many other countries are absolutely demonstrative of its practical efficiency. In France, from 1882 to 1886, about 1,150,000 sheep and 110,000 oxen

give better results; they have, besides, the advantage of requiring but one operation. When inoculation of anthrax will have been sufficiently improved it may render great services from the standpoint of prophylaxis of anthrax in the ox.

Since 1882 Oemler has each year practised Pasteur's inoculation in the domain of Pakisch (Prussia). The results have not been satisfactory either for cattle or for sheep. In the former the mortality by anthrax has been on an average 1.4 per cent.; in the others 4.2 per cent. Some of the animals which died had been inoculated several times.

BIBLIOGRAPHY. The publications relating to bacterian anthrax are very numerous. We are not able to make a complete enumeration of them here. For special literature previous to 1850 we refer the reader to Heusinger's work,¹ in which he records three hundred and sixty-two memoirs or writings, and for the articles appearing from 1850 to 1884 to the works of W. Koch,² wherein he reviews the bibliography of diseases of anthrax in man and animals.

ANTHRAX IN THE VARIOUS DOMESTIC SPECIES.

I. Anthrax of the Ox.

Symptoms. In the ox the *acute form*, without external localizations, is the most frequent. It is marked from the onset by

were inoculated; for the sheep the total loss was 1 per cent. instead of 10 per cent. (average mortality of previous years); for the oxen the mortality, which was formerly 5 per cent., had fallen to 0.5 per cent.

In 1887 anthrax was observed in 1248 oxen, 2858 sheep, and 25 horses; these animals represented a value of \$76,400. According to the reports of the veterinary board of health, inoculation has been practised upon 6403 bovines and 28,234 sheep. The mortality has been very small: 10 oxen (0.15 per cent.) and 146 sheep (0.52 per cent.). They have mentioned in man eleven cases of malignant pustule, four of which ended fatally (Tisserand: Rapport sur le service des epizooties eu 1887 in Recueil vét., 1889).

In Hungary, in 1889, out of 3279 inoculated oxen the mortality by anthrax was 0.27 per cent.; during the preceding years it varied from 6 to 12 per cent. For 22,767 sheep the losses were 2.18 per cent. instead of 10 per cent., a figure which was recorded in previous years. In 1890 they inoculated 21,289 oxen and 190,229 sheep. In the former the mortality by anthrax was 0.37 per cent., and in sheep 2 per cent. When performed in infected flocks inoculation has arrested the epizootic. It may be practised without taking into account the breed, sex, or age. (Hutyra: Jahresbr. über das veterinärwesen in Ungarn, Budapest, 1891.)—N. D. T.

¹ Die Milzbrandkrankheiten der Thiere und des Menschen, Erlangen, 1850.

² Milzbrand. u. Rauschbrand Deutsche Chirurgie von Billroth und Lüke, 1886.

intense fever; the temperature reaches 41° – 42° C.; it is distributed irregularly on the superficial regions; the hair is erect; the pulse accelerated (80 to 100 pulsations and more), small, imperceptible. The mucous membranes of the head are red and often cyanotic, the conjunctiva is much infiltrated, and ordinarily shows petechiæ; sometimes there is weeping. Weakness and stupefaction are very pronounced, inappetence is complete, and rumination has disappeared; the gait is uncertain; trembling is observed upon the whole surface of the body, especially upon the flanks and the hind-quarters. The patients remain behind the herd, their eye is dull, and their expression is dejected. There are cases where the clinical tableau is that of a serious cerebral affection. Sometimes stupefaction is replaced by rabiform attacks; the patients bellow, scratch the ground, mount into the manger, push against the wall, etc. A serious dyspnœa, independent of any important pulmonary lesion, may also dominate the scene. Gastric troubles: constipation, slight tympanites, colics, diarrhea, expulsion of bloody matter, etc., are also common manifestations of anthrax in the ox. The urine often contains blood (hematuria). Impregnated females abort or show symptoms which indicate this accident. The natural orifices (mouth, nostrils, eyes, anus, vagina) give exit to liquids mixed with blood. Generally stupefaction, coma, and weakness continue to increase, and death follows in convulsions within from twelve to forty-eight hours.

The cases of *hyperacute anthrax* (*Anthrax acutissimus*) are noticed in strong animals and in the beginning of epizootics. The animals die suddenly or within a few hours, showing all the symptoms of cerebral apoplexia or intoxication. Sometimes, on opening the stable, we find animals dead which were in perfect health the day before.

The cases of *anthrax with subacute evolution* are less common. This form has a duration which varies from three to seven days; this latter term is rarely exceeded. It is characterized by fever, considerable emaciation, and periodical remissions.

Anthrax tumors are observed in the ox, sometimes as primary accidents, at other times during the course of acute or subacute anthrax. They appear upon the head, neck, chest, shoulders, abdomen, sheath, mammæ, flanks, and extremities. Ordinarily isolated, and more or less limited or diffuse, they are not very sensitive, and may be painless in the beginning; their shade is bluish

or dark red; upon section they have a lardaceous, gelatiniform consistency; they never contain pus, but are often sphacelent; in this latter case the skin which covers them is affected by gangrene. The lesions of anthrax of the buccal mucous membrane (gloss-anthrax, palato-anthrax) present the appearance of vesicles or noduli more or less voluminous, and are developed upon the tongue, lips, cheeks, and palate; they produce abundant salivation, and sometimes dyspnoea. Anthrax tumors of the rectum (*sang de lombes*) occasion diffuse tumefaction of the mucous membrane of this reservoir and a bloody discharge; they determine violent expulsive efforts, sometimes followed by anal prolapsus. The cause of these lesions is generally recognized to be the manœuvres performed in practising rectal exploration.

Differential diagnosis. The diseases of the ox which are ordinarily confounded with anthrax are: poisonings, encephalitis, cerebral apoplexia, pulmonary congestion, sunstroke, hydrophobia, gastro-enteritis, and symptomatic anthrax. When the infection has a very rapid course the diagnosis is often only established at the autopsy. It is proven by finding the bacteridium and by the results given by inoculation (see Differential Diagnosis of Symptomatic Anthrax).

II. Anthrax of the Horse.

Pathology. In the equine species all the observations mentioned under the name of "anthrax" cannot be accepted without reserve. Many of them refer to petechial fever.

In solipedes we usually find anthrax in the localities where it exists in an enzootic state, and where, besides the ox, all domestic animals may become infected. Its ordinary cause is the ingestion of spores or bacteridia deposited in the food. Infection may also take place by fly-bites or by the use of a harness manufactured of leather from hides coming from animals affected by anthrax (Bobertag).

Symptoms. The *acute* and *subacute* are the most common. Among the first symptoms we observe a high fever of infectious (39.5° to 41.5° C.); an accelerated pulse, which is small, then imperceptible (80 to 120 pulsations per minute). Very often hyperthermia is accompanied by chills and convulsive contractions of the muscles; the external temperature is distributed irregularly. The conjunctiva and other mucous membranes of the head are cyanotic;

sometimes they have a yellowish reflex ; we often see bilateral weeping. The psychic condition is greatly affected ; the expression is sad, without intelligence ; the patients are apathetic, stupefied-like, and the gait is staggering ; in some cases we observe symptoms of cerebral excitement, which simulate encephalitis (excitement, restlessness, and spasms). Colics are constant and often premature ; rarely violent ; they are accompanied by a liquid bloody diarrhea ; we may observe vomiting (Burke). Respiration is accelerated and laborious. When the mucous membrane of the posterior part of the mouth is the seat of a specific tumefaction we observe the symptoms of angina (salivation, dysphagia, considerable tumefaction of the laryngeal region, intense dyspnoea, and sometimes asphyxia). Death, which is indicated by profuse perspiration, occurs within from six to thirty hours. Recovery is rare.

Cutaneous tumors of anthrax are ordinarily developed upon the belly, chest, the inner fascia of the legs, the scrotum, the vulva, etc. ; those which are located upon the posterior legs result in lameness. This form has a somewhat slower course than the preceding. Its average duration is from two to three days.

Gloss-anthrax has only been found exceptionally in the horse (Gresswell).

In India, Burke has observed in the horse a particular form of *anthrax with remittent course*. At certain moments the temperature was below the normal, all the morbid phenomena disappeared, and the patients seemed to have recovered ; then, within a few days, fever reappeared and the animals died of exhaustion. The clinical symptoms described by Burke recall those of intermittent fever of man. Among the characteristic symptoms he mentions rapid emaciation, beginning a few hours after the first attack, and paralysis of certain muscular groups.

Differential diagnosis. Anthrax may be recognized by its tumultuous and febrile evolution, by the diversity and inconstancy of its symptoms, which show nothing regular in their appearance. Affections which most often lead to mistakes in diagnosis are : petechial fever, colics, cerebral apoplexia, pulmonary oedema, and septicemia. In the chapter on Petechial Fever we have pointed out that formerly this disease was erroneously considered as a form of anthrax : in petechial fever, in fact, the bacteridium is wanting ; authors have never produced anthrax by inoculating this affection ; and not one case has ever been mentioned which establishes its

contagiousness. The diagnosis is based upon the detection of the bacteridium and upon the results of the inoculation in the rabbit or the sheep.

III. Anthrax of the Sheep.

Pathology. It is also designated under the names of "disease of the blood," "blood-stroke," "spleen blood." Anthrax of the sheep has been specifically recognized by Gerlach. In general, the animals become infected in eating food soiled by bacteridium or its spores; exceptionally the inoculation is caused by fly-stings, wounds of the skin at the time of shearing (Nocard), etc.

Symptoms. *Apoplectiform anthrax* (*Anthrax acutissimus*) is the most frequent in the sheep. The affected subjects suddenly present the symptoms of apoplexia; they stagger, fall, and are taken with spasms, convulsions; black blood escapes by the natural openings. Death occurs in a few minutes. Often, in the morning, on opening the sheepfold one or several sheep may be found dead.

Acute anthrax has a somewhat longer duration (from half an hour to two hours). Sometimes it is marked by the symptoms of cerebral hyperemia: excitement, stamping, uncertain gait, etc.; in other instances by pulmonary congestion; very accelerated respiration and circulation, cardiac palpitations, cyanosis of the mucous membranes, bloody urine, bloody discharge by the natural orifices, etc.

Subacute anthrax, which is very rare, is almost always preceded by certain prodromes, particularly by digestive troubles. These symptoms are those of acute intestinal phlegmasia. We especially observe constant expulsive efforts and continual movements of the tail.

In some cases we observe *anthrax tumors* upon the head, throat, and mammæ.

Differential diagnosis. In the sheep anthrax has frequently been confounded with *malignant œdema*. The diseases designated under the name of "swine plague," "fire," "flying gangrene," and described by Haubner and others as forms of anthrax characterized by crepitant tumefactions of the posterior members, belong certainly in the domain of malignant œdema (see vol. ii. p. 315).

Anthrax of the goat much resembles that of the sheep; its course is a little less rapid.

In the countries of the Nord, in Iceland, and the Faroe Islands,

they call *Bradsot* an acute infectious and very malignant disease of the sheep, a disease which ends in death within a few minutes or hours. Its principal symptoms are inappetence, colics, and a foamy saliva which runs out of the mouth. At the autopsy we find hemorrhages in the abomasum. Viborg considered *Bradsot* an anthrax affection. Tousson has recognized that it has nothing in common with anthrax; it is not transmitted to subjects of other animal species.

IV. Anthrax of the Pig.

Pathology. Authentic observations of spontaneous anthrax of the pig are very rare. Bacillary swine plague, which is so often confounded with anthrax, has nothing in common with this affection. At the present day we know that the pig possesses almost complete immunity from anthrax, and that it is very difficult to infect it (experiments of Brauell, Renault, Toussaint, Arloing, Cornevin and Thomas, etc.). However, at the periods when anthrax makes numerous victims in countries where it is stationary, the pigs, especially those which ingest anthrax meat, may become affected. Inoculation takes place usually by the buccal or pharyngeal mucous membrane.

Symptoms. Anthrax tumors of laryngeal and pharyngeal cavities determine symptoms of angina. The fever is intense; we observe considerable tumefaction in the submaxillary glands which may extend along the trachea as far as the chest and to the inner fascia of the anterior legs; we observe, besides, ptyalism, movements of regurgitation, dysphagia, nausea, vomiting, cyanosis of the buccal mucous membrane, a dyspnoëic wheezing, rattling respiration, etc. Death occurs, as a rule, by asphyxia.

In the beginning of gloss-anthrax and palato-anthrax we see upon the mucous membrane of the tongue, palate, lips, etc., the development of vesicles which have a clear shade and soon become violet, then black. Zschokke has observed furunculus upon the dorsal region.

V. Anthrax of the Dog.

Our knowledge of anthrax of the dog is as yet very imperfect. Infection is almost always produced by ingestion of anthrax meat. We observe ordinarily the intestinal form or buccal and pharyngeal localizations. Out of seven dogs which had feasted upon the carcass of a cow that had died from anthrax, five succumbed the same

evening (Cornevin). Peuch made the autopsy of a dog which had been infected through a wound on the point of the tongue by eating anthrax meat. It is probable that several of the facts which are mentioned under the title of "anthrax of the dog" were but cases of mycotic enteritis (intoxication by ptomaines). In man also this latter affection (*Mycosis intestinalis*) was formerly generally confounded with anthrax.

VI. Anthrax of Gallinaceæ.

Pathology. According to Pasteur, poultry are refractory to anthrax, because the temperature of their blood is too high. By the action of cold we may destroy this immunity and infect them fatally.

These statements are weakened by numerous positive facts which have resulted from experimental researches made by Feser and Oemler, and by negative results obtained by employing cold (Feser and Koch). In his experiments Oemler has observed that the small breeds of birds (sparrows, redbreasts, chaffinch, canary, goldfinch, ortolan, etc.), and, in general, all young birds, take anthrax easily; birds of large breeds are less sensitive to it; as for birds of prey, they possess complete immunity. Poultry ordinarily become infected only during the course of epizootics, when they ingest blood or meat of anthrax cadavers. Various domestic birds may become infected under such conditions (chicken, goose, duck).¹

¹ In ordinary conditions the chickens are truly refractory to anthrax. Koch, Gaffkey, Löffler, Perroncito, Kitt, and Hess have only obtained negative results. It is probable that Oemler had to deal with malignant œdema and not with anthrax. In 1878 Pasteur recognized that the chicken dies from inoculated anthrax if its temperature is lowered by placing it in a vertical position upon a board and dipping the lower third of the body into water at 25°. Colin, Feser, Koch, etc., have failed in their experiments, because they did not realize the conditions which insure sufficient reduction of temperature in order to permit active bacteridian increase, and not fatal by itself. Wagner, who has taken up this question, has come to the following conclusions:

"1. In ordinary conditions the chicken is refractory to anthrax. Immunity is due to phagocytic activity of the leucocytes.

"2. The bacteridium may become developed and preserve its virulence in the body of the chicken; its inoculation is not an indifferent phenomenon; it is accompanied by febrile reaction.

"3. The chicken may be affected by anthrax, and die from it when it is deprived of the salutary assistance of leucocytes. These conditions are established: *a*, best by cooling of the chicken in a cold bath, circumstances under which all chickens die (six in six); *b*, by the action of antipyrine, which gives a smaller mortality (six in eleven); *c*, by chloral, the action of which is still less marked." (Wagner: *Annales de l'Institut Pasteur*, 1890.)—N. D. T.

Symptoms. In poultry anthrax has a very rapid, tumultuous course. Death may occur suddenly or within a few hours ; often the subjects fall from their perches, are overcome by trembling, and die in convulsions within a few moments ; bloody liquids escape from the mouth, nostrils, and anus. In other cases the duration of the disease is about twenty-four hours ; the patients are weak and their feathers are erect, the wings are hanging, the mucous membranes and the comb are cyanotic ; we observe dyspnoea and bloody diarrhea. In others, also, we notice anthrax tumors upon the comb, the maxillary lobules (wattles), upon the conjunctiva, the tongue, palate, extremities, and the interdigital membranes, etc. We may confound anthrax of chickens with cholera and cerebral apoplexia.

In man anthrax has as a starting-point an integumentary wound (meat-cutters, butchers, tanners, shepherds, veterinarians), which is marked at first by a *malignant pustule*, and which appears ordinarily upon the hand, the arm, face, and neck ; then, secondarily, by anthrax fever. In most cases the process terminates in death. Intestinal anthrax is produced by the consumption of infected meats.

The treatment of malignant pustules comprises scarification, cauterization of the tumor, and the use of energetic disinfectants (sublimite, phenic acid).

HYDROPHOBIA (LYSSA: RABIES).

A. Generalities Upon Hydrophobia.

HISTORY. Rabies (hydrophobia, *lyssa*, *rabies*) has been known from the most ancient times. Four centuries before Christ Aristotle described it, and indicates its transmissibility in these terms : "Dogs suffer from hydrophobia, which provokes in them a state of madness ; all the animals bitten by dogs affected by it become rabid in the end." In the works of Virgil, Horace, Ovid, and Plutarch we find notes relating to this affection. In the first century of our era Celsus mentions human rabies and calls it hydrophobia ; Dioscorid recommends excision of the tissues upon the surface of the bite. Galen (second century) advised special remedies against rabies. Among the ancient authors who have referred to it we must also mention Pliny (the younger), Columelle, and

C. Aurelianus. Nothing is known of rabies during the obscure and long period of the middle ages. In 1591 Bauhin¹ mentioned a case of transmission of rabies from the wolf to man. In 1604 hydrophobia existed in an enzootic state in Paris (Audry). Under this form it prevailed in Italy toward the end of the twelfth century (Baglio, Ramazzini); during the year 1708 in Suavia;² from 1719 to 1723 in France and Germany; from 1754 to 1760 in England; from 1779 to 1807 in America, especially in the West Indies and in Peru. Toward the end of the last century and in the beginning of the present it prevailed all over Europe, and numerous rewards were offered for the discovery of means of curing it. Among the investigators who expressed the most correct ideas upon its character we must particularly mention Chabert and Hunter. From 1803 to 1830 it destroyed the foxes of southern Germany and Switzerland (Köchlin and Franke). In 1814 and 1815 Viborg (of Copenhagen) and Waldinger (of Vienna) made the first experimental researches upon hydrophobia. In England Delabère-Blaine and Greve (1817-18) gave a good clinical description of it. In 1822 it existed in Holland; in 1823 and 1824 in Berlin; in 1824 in Sweden and in Russia; from 1823 to 1830 in Germany. In 1828 Hertwig published his *Contributions on the Study of Hydrophobia*, a work in which he relates a long series of attempts at transmission which have increased our knowledge of the disease. At the same period it was studied by Youatt (1830) and Prinz (1832). In the years which followed 1830, and in 1852-53, it claimed numerous victims in Prussia, and from 1838 to 1843 in Austria and Würtemberg. In 1853 at the hospital of the veterinary school of Berlin 150 cases were observed in the dog. At Hamburg in the same year 267 were recorded.

In 1861 hydrophobia existed in an epizootic state in the countries of the Rhine and in France; from 1863 to 1871 in Würtemberg, where the number of rabid animals amounted to 597 (449 men were bitten; 23 of these died); from 1862 to 1867, and 1873 to 1876, in Vienna; in 1865-66 in Saxony; from 1871 to 1876 in Saxony, Bavaria, and Prussia. Since the promulgation of the law on contagious diseases in Germany the number of cases of hydrophobia has considerably diminished.

Up to modern times our knowledge of this disease has been

¹ Bauhin: *Memorabilis historia luporum aliquot rabidorum*.

² Camerarius and Scharff: *Dissertatio inogural de Alyssso clave*, Tübingen.

vague and erroneous. Notwithstanding that its infectious character was demonstrated for a long time, the possibility of its spontaneous development was generally admitted. Among the influences which were generally considered as capable of producing it we especially mention great heat, unsatisfied venereal desires, deprivation of drinking-water, nervous excitement, anger, jealousy, rich, intensive food, etc. Since 1854 Virchow combated this etiological opinion. The infectious nature of hydrophobia has only been admitted within the last ten years. In 1881 its domain, which had been obscure up to that time, was distinctly cleared by Pasteur, whose works have led to the discovery of a method of preventive inoculation.

Etiology. Numerous experimenters (Raynaud, Lannelongue, Gibier, Fol, Babes, Pasteur, Koch, and others) have vainly attempted to isolate the infectious agent of hydrophobia. But Pasteur has recognized that it exists in a pure state in the central nervous system (brain and spinal cord) of diseased animals; it is also contained in the salivary and lachrymal glands, in the pancreas, the mammæ, and in the products of these organs. In the secretions it is mixed with various micro-organisms. The blood itself does not seem to contain it.¹ The virulent germ of rabies is *fixed* and *endogenous*. Paul Bert has shown that this germ is indeed a typified element: when filtered through plaster disks rabid saliva has never been found virulent. By making a microscopical examination of the cerebral matter of rabid animals Pasteur has seen numerous very fine corpuscles, which fix the aniline colors and present them under the form of very small points; according to this scientist, we have to deal with micro-organisms which are neither micrococci nor bacilli. All attempts at cultures which he has made have been unsuccessful. We have learned from recent researches of Babes that by means of modern bacteriological methods we are not able to discover in rabid tissues constant and characteristic micro-organisms. According to this author, there exists in the central nervous

¹ Rabid virulence has also been observed in the upper renal capsules, in urine, spermatic fluid, and lymph. It has been demonstrated that the blood is never virulent, which is contrary to what has been for a long time admitted. The possibility of intraplacental transmission of hydrophobia seems to be established by several observed facts and by a few experimental results. The researches of Nocard and Roux have shown that the virulence of the saliva of the dog precedes by one, two, and sometimes three days the appearance of the first rabid symptoms. (Annal. de l'Institut Pasteur et Bullet. Soc. cent. Vét., 1890.)—N. D. T.

system of rabid animals an element with unknown morphological characters, but which may be cultivated in series, and preserves the property of generating hydrophobia; if subjected to the influence of external agents, it behaves in the same manner as most micro-organisms; it is much more resistant to the action of phenic acid than any known microbe. Babes' culture produces hydrophobia in the dog, rabbit, guinea-pig, cat, rat, and mouse when it is inoculated either in the eye or in the meninges after trephination.

The existence of the rabid germ seems to be much greater than was formerly admitted. In the experiments of Hertwig the inoculations of saliva and of blood of hydrophobic animals, which were made twenty-four hours after death, have constantly given negative results. But saliva which was obtained within twenty-four hours has been found infectious by Gibier, and Mergel has recognized the virulence of the brain fifteen days after death. Similar results have been obtained at Dorpat. Pasteur submitted the encephalon of rabid dogs to the action of cold (temperature of 12° C.) for three weeks, without loss by the nervous substance of its noxious property; this has been preserved for months in brain matter which was enveloped in moistened phenicated gauze. During the summer nervous tissue preserved in hermetically-sealed tubes has retained its activity for three or four weeks. Pasteur has recognized that, in ordinary conditions, the virulence of rabid matter disappears only after four to five days, when putrefaction is already fairly well advanced. Formerly it was believed that it disappeared as soon as the bodies were cold, that is to say in the twenty-four hours following death.

The transmission of the infectious agent occurs directly; no authentic fact exists of contamination by any intermediary agent. Almost always, not to say constantly, infection is produced by bites. Hydrophobia must thus be considered as a "disease of inoculation." Galtier admits the possibility of infection by the digestive passages, by ingestion of saliva, milk, or meat coming from a rabid animal; but if this author has in reality produced experimental hydrophobia by making the rabbit swallow rabid saliva, no fact has as yet been mentioned which establishes that ingestion of rabid milk or meat has produced it.¹ Intra-cranial

¹ Nocard has never succeeded in transmitting hydrophobia by the digestive tract even when the animals several times ingested considerable quantities of virulent

inoculation of rabid milk has given positive results (Nocard, Roux, Bardach). The question of heredity of hydrophobia is yet under investigation. Perroncito and Carito have succeeded in infecting a guinea-pig by inoculation of the spinal marrow of a young rabbit which was the offspring of a rabid mother. Callignac and Gibier have mentioned cases of heredity of the disease. The experiments of Renault, Roux, and others have given but negative results.

Celli has studied rabid contagion from its standpoint of resistance to external agents. The virulence is destroyed within half an hour by steam; in one hour by a temperature of 50° to 60° C.; in twenty-four hours by a temperature of 45° C., by solutions of sublimate (1 per 100,000), of permanganate of potash (2 to 5 per cent.), and by alcohol (50 to 90 per cent.); in five days by alcohol at 25 per cent., and in seven days by alcohol at 15 per cent. The emulsion of cerebral substance loses its virulence if we acidulate it with one or two drops of acetic acid, or if it is rendered slightly alkaline with soda.

Pathology. When the rabid virus is introduced under the skin by a bite it may remain upon the surface of the wound for some time and penetrate more or less rapidly into the organism by the nerves and vascular canals.¹ The brain and spinal substance seem

nervous matter. Among the experiments which he has made upon this subject the following is particularly interesting: Within two months a young fox had eaten, without becoming infected, the brain and spinal cord of twelve rabid dogs: he was, however, not refractory and had not acquired immunity, for later he died from hydrophobia which was inoculated by trephination. (See Bull. Soc. Cent. Vét., 1884.)—
N. D. T.

¹ The course followed by the rabid virus, in order to enter the organs where it has been found, is not as yet determined exactly. We know, however, that it progresses especially in the nervous cords and along the cerebro-spinal axis. The method of propagation of the virus which was observed by Duboné (of Pau) has been the object of recent experimental researches. By inoculating hydrophobia into the thickness of the sciatic nerve, Vestea and Zagari have seen the development of a paraplegia which was more marked on the side where the inoculation had been made, and invaded the body from backward forward; the lumbar cord was virulent before the medulla oblongata. By making the inoculation into the median nerve, paralysis progresses from forward backward, and the medulla oblongata was virulent before the lumbar cord. By inserting the rabid matter into a hind leg after a section of the cord, this was only found to be virulent in the posterior part of the section. Like most other virus, that of rabies seems to exercise its noxious action by the toxic matter which it generates. By injecting into dogs the product obtained by the filtration of an emulsion of rabid spinal substance, De Blasi and Travali have produced an intoxication and paralytic phenomena without communicating hydrophobia.

to be the most favorable soil for its development. When it pullulates, especially in the brain, it generates the *furious form of rabies*; if it invades first the spinal cord, it produces the *dull or paralytic form of the affection* (Pasteur).¹ The experiments of this author have demonstrated that the period of incubation is reduced to its minimum when the virus is brought directly upon the encephalon. Success is far from being always certain.

The duration of incubation is longer in rabies than in other infectious diseases; in the dog it is from three to six weeks on an average; sometimes it lasts only a few days; it may also be prolonged for months. These differences depend probably upon the slowness or rapidity of the propagation of the virus in the organism and the time it remains in the wound.

AFFECTED ANIMALS. We may observe hydrophobia in the following species: dog, cat, ox, donkey, mule, hinny, sheep, goat, hen, pigeon, wolf, fox, prairie dog, hyena, badger, marten, monkey, stag, deer, antelope, skunk, rabbit, guinea-pig, rat, mouse. Man is also frequently the victim of it.

In the large majority of cases it is observed in the dog. The predisposing causes which were advanced formerly: breed, age, sex, climate, country, have no etiological influence. Its frequency varies with the seasons. It always kills more subjects in summer than in winter.² There are animals which seem to possess a certain

The nervous centres of animals which showed these troubles were not virulent. (Roger: *Traité de Médecine*.)—N. D. T.

[The experiments of Peyraud with oil of tansy, which determines rabiform symptoms, corroborate this opinion. They deserve another trial.]—L. T.

¹ At its late period the furious form of rabies is accompanied by paralytic phenomena, but in the so-called *dull or paralytic* form—which is but a clinical variation of the infection—premature *akinesis* appears in the various regions, upon the masseters, certain muscular groups, upon one or several members, and the posterior quarters.

Intra-cranial and intra-ocular inoculations almost always produce *furious hydrophobia*. On the contrary, by injecting the virus into the subcutaneous connective tissue or into a vein, we generally produce the *paralytic* form; in a certain number of cases, however, we may obtain the first form by using small quantities of virus. The smaller the quantity of virus that is injected into the subcutaneous connective tissue or into a vein, the more easily furious hydrophobia is obtained (Pasteur). The hypodermic inoculation gives more numerous positive results in emaciated animals than in those the integument of which is lined with a thick adipose layer (Helman). Intra-muscular inoculations succeed better than those made into the connective tissue.

—N. D. T.

² This theory, which is generally admitted, is weakened by statistics. Bourrel has already shown, according to his record of the cases of hydrophobia which were recog-

individual immunity. Rabies is relatively common in large cities, because dogs are ordinarily very numerous and the measures of sanitary police very badly enforced.

Hydrophobia seems stationary in regions where wolves exist in large numbers (Vosges, Carpath Mountains, and in Russia, etc.); it is kept in existence by these animals by transmission of the disease from the wolf to the dog, and reciprocally.

STATISTICS. In the German Empire, in 1886, the number of animals affected by rabies was 578 (438 dogs, 3 cats, 5 horses, 92 oxen, 32 sheep, 7 pigs, 1 goat). The districts of Gumbinnen, Bromberg, Oppeln (Heydekrug, Schroda, Lyck, Inovorazlaw, Osterode), upon the Russo-Polish frontier have furnished the greatest number of these. In Austria, during the same year, the number of cases of rabies was 858 (769 dogs). In 1887, in France, 1643 cases were seen in the dog; in Belgium, 144; in Holland, 69.¹

nized in his infirmary from 1859 to 1872, that they were not more frequent in summer than in winter. 314 cases of rabies of the dog observed at the Alfort school during the last four years are divided as follows for every three months:

	1887	1888	1889	1890
January, February, March	22	60	33	15
April, May, June	17	25	14	4
July, August, September	20	15	10	5
October, November, December	21	30	17	6

—N. D. T.

¹ In France during the year 1887 hydrophobia was recognized in 2567 dogs or cats and in 426 animals of other domestic species; these latter represent a value of about \$20,000. It has appeared in all departments: in 31, less than 10 have been found; in 17, from 10 to 20; in 26, from 20 to 50; in 9, from 50 to 100; and in 2 more than 100 (Basses-Pyrénées, 101; Seine, 644). In the Department of the Seine, from 1883 to 1888, the number of cases of hydrophobia increased every year; 1883, 182 cases; 1884, 301; 1885, 518; 1886, 604; 1887, 644; first half of the year 1888, 500. In 1887 the Board of Health reported 668 cases of persons who were bitten, 22 of whom died (3.33 per cent.). The same year 1431 Frenchmen who were bitten by mad or suspected dogs presented themselves at the Pasteur Institute in order to undergo the prophylactic treatment; 16 died from rabies (1.11 per cent.). In the Department of the Seine alone 530 persons were bitten by rabid animals; out of 306 which had undergone Pasteur's inoculation, 2 died from hydrophobia (0.76 per cent.); of 44 which were not treated 7 died of rabies (15.90 per cent.). (Tisserand: Rapport sur le service des épi-zooties en 1887.) At the Alfort school, during the year 1887 they recognized 80 cases of rabies in the dog; furious hydrophobia, 38 cases; paralytic form, 22 cases; rabies which were recognized at the autopsy (in doubtful cases where inoculation had been made), 22. Here are the figures for the three following years: 1888, 130 cases. Furious rabies, 53; paralytic rabies, 36; rabies which were recognized at the autopsy, 41. 1889, 74 cases. Furious rabies, 37; paralytic rabies, 19; rabies which were recognized at the autopsy, 18. 1890, 30 cases. Furious rabies, 8; paralytic rabies, 7; rabies which were recognized at the autopsy, 15.—N. D. T.

Anatomical alterations. The alterations found at the autopsy of rabid animals are neither constant nor specific: Hydrophobia is especially indicated by the absence of important organic lesions. We mostly observe alterations that are but little marked. The cadavers are very emaciated and rapidly undergo putrid decomposition; in the large species the abdomen swells rapidly. The blood is thick, hardly coagulated, and of blackish-red color. The muscles seem to have undergone granulo-fatty retrogression. The heart, liver, and kidneys are affected by parenchymatous fatty degeneration. The buccal mucous membrane, especially that of the base of the tongue, is red and inflamed; the tonsils are hypertrophied, infiltrated, phlogosed; the salivary glands are hyperemic. The laryngeal and pharyngeal mucous membranes are red, tumefied, and often overrun by small hemorrhagic centres. In the pharynx and œsophagus we sometimes find various bodies. In the stomach, which contains little or no food, we find accumulations of foreign or indigestible matter which may be in considerable quantity: straw, hair, feathers, pebbles, pieces of brick, wood, leather, pieces of whip-handles, etc. Wortley Axe, in a total of 200 autopsies, has found 180 times (90 per cent.) the absence of food and the presence of indigestible foreign bodies in the stomach. For him this latter fact is the most important from a diagnostic standpoint.¹ The gastric mucous membrane is inflamed; upon its folds we frequently observe petechiæ and hemorrhagic erosions. The intestine is generally empty; its mucous membrane is hyperemic and obstructed; the same is the case with the mesenteric ganglions. The spleen is congested and sometimes contains small hemorrhagic centres.

In the urine we find albumin and coloring matter of the bile (in the dog).²

The respiratory mucous membrane is tumefied and bluish; the lungs are congested.

¹ The post-mortem diagnosis can only be established with certainty by inoculation; but when at the autopsy of a dog which has manifested aggressive tendencies during the last period of life, or which has bitten animals or people, we recognize the ordinary symptoms of rabies, especially the presence of foreign bodies in the stomach, we must, without hesitation, affirm the case, and proceed accordingly. Many authors admit that *intestinal obstruction* in the dog usually produces rabiform manifestations and a tendency to biting. We persist in thinking that these authors take the exception for the rule.—N. D. T.

² The urine passed by the rabid man quite frequently contains albumin and sugar. The presence of sugar in the urine of rabid animals has been mentioned by Nocard and Roux.—N. D. T.

The encephalon and spinal cord offer very inconstant alterations: at times we notice hyperemia, œdema, punctiform hemorrhage; in other instances no other macroscopical lesion can be found. According to Csokor, at the microscopical examination we often observe an accumulation of leucocytes along the dilated capillaries, in the walls of the small vessels, and particularly in the gray substance, which is marked besides by microscopic hemorrhages.¹

Prognosis. In his experiments Pasteur has seen some animals recover after the appearance of the first rabid symptoms. Besides these observations no authentic case exists of recovery of rabies, and at the present time it must be considered as absolutely fatal. The same is the case with man; the death-rate of human hydrophobia is 100 per cent.

The number in cases of rabies produced by inoculation is much less. In Hertwig's experiments it was 37 per cent.; in those of Renault, 67 per cent. These different results depend upon the details of the operation: often the virus does not penetrate into the wound or it is removed by the hemorrhage. We explain in the same manner the fact that all bitten animals do not become affected. The disease is developed in about 20 or 30 per cent. of cases of inoculation by bites (Hertwig gives an average of 5 per cent.; Haubner, 40 per cent.; the Alfort School, 33 per cent.; the Lyons School, 20 per cent.).

In man hydrophobia occurs in 8 to 47 per cent. of cases of infection by bites (Bollinger); according to Pasteur, this proportion varies from 16 to 80 per cent. According to Bollinger, when cauterization is not performed it reaches 83 per cent.; in the contrary condition it amounts to 33 per cent.

Treatment. The curative treatment of hydrophobia is illusory. We may only resort to prophylaxis in order to obtain any result. In animals cauterization of the wound by means of the hot iron,

¹ In the nervous centres the microscope shows also numerous small bloodvessels, which are obstructed by hyalin thrombi; coagulum of the same nature fill the perivascular lymphatic sheaths, to which they give a moniliform aspect. In these sheaths we find also an abundant accumulation of leucocytes. In the surface of the bulbar nuclei and the anterior ends of the spinal cord the nerve-cells are either surrounded by leucocytes, or partially destroyed by these, or undergo fatty degeneration. The white substance of the spinal cord and the nerves have undergone alterations involving the myelin and the cylindrax (Roger, loc. cit.). The accumulation of white corpuscles in the perivascular lymphatic sheaths—which were once considered as a pathognomonic lesion of rabies—is an anatomo-pathological character common to various infectious diseases with encephalic symptoms.—N. D. T.

caustic potash, sulphuric acid, sublimate, etc., or amputation of the bitten region is impracticable in most cases. Measures of sanitary police and a tax upon dogs are the most successful means to prevent hydrophobia. In Bavaria statistics show that it has considerably diminished since the establishment of the tax on dogs. In cities the use of a muzzle is also a very efficient measure, provided the apparatus answers all requirements. In Berlin the muzzle has given excellent results.

In Bavaria (where the tax on dogs dates from 1876 and the laws on contagious diseases from 1880) the number of cases of rabies has been reduced from 812 in 1873 to 11 in 1885; in Baden, from 53 in 1874, it has fallen to 0 in 1885; in Saxony, from 287 in 1866, to 16 in 1885; in Prussia (where the law upon contagious diseases acts alone), from 672 in 1878, to 352 in 1885.

INOCULATION. In 1884 Pasteur recognized that the rabid virus becomes gradually weaker when it is inoculated from the dog to the monkey, and when cultivated in series upon this latter animal. The virus which is attenuated in this manner no longer produces hydrophobia when it is injected under the skin of the dog, and, by repeated inoculations, the animal acquires immunity. More recently Pasteur has made known another process of inoculation. He uses dried-up substance of the spine, the virulence of which is attenuated by the following method: In a rabbit which has died from rabies transmitted by inoculation of very virulent virus (*fixed virus*), they remove the spinal cord and cut it into shreds of the length of six centimetres, these are then suspended in bottles, by means of threads; these receptacles must contain a layer of caustic potash of 1 centimetre thickness, and their openings should be closed with a tampon of wadding. These shreds of rabid spinal cord are subjected to a steady temperature of 20° C. Within three or four days they are dried and form strips which may easily be pulverized. Their virulence is completely destroyed within a certain time (fourteen days). When the rabbit is inoculated with *very virulent* rabid spinal matter which has been dried from twenty-four to forty-eight hours, hydrophobia is manifest in seven days; it appears only after eight days when desiccation has been prolonged from three to five days, and within fifteen days when it has been continued from six to nine days. In order to give the animals immunity from rabies, they begin to inject spinal cord which has its virulence much weakened; then we make successive inocu-

lations with substances which are in a less dried state, and finally with fresh virulent matter. These inoculations may produce in subjects experimented upon transient rabid phenomena. According to Babes, eight to fifteen days after inoculation the dog often shows depression, inappetence, emaciation, nervous troubles, a tendency to biting, and even paresis of the front legs; but these symptoms disappear rapidly.

Pasteur has observed that the virulence increases gradually and that the duration of the period of incubation diminishes when hydrophobia is inoculated in series to rabbits or to guinea-pigs. In order to obtain the most *virulent virus* (*fixed virus*), he proceeds in the following manner: he trephines the skull of the rabbit with a crown 6 millimetres in diameter, and by means of a Pravaz syringe fitted with a needle which is bent at a right angle he injects under the meninges a small quantity of nervous matter coming from a dog which had died from hydrophobia (by injecting the virus under the skin the period of incubation is much longer); within fifteen days hydrophobia appears upon the inoculated rabbit. By continuing the inoculations from rabbit to rabbit, the period of incubation becomes gradually shorter; upon the twenty-fifth subject, it is eight days; after the fiftieth, it is only seven days. At this time the maximum of virulence has been reached. By means of inoculation of ordinary fresh virus (street rabies) the period of incubation is from fifteen to sixteen days.

These discoveries of Pasteur have been tested and entirely confirmed by a French commission, in which appeared the celebrated names: P. Bert, H. Bouley, Tisserand, Villemin, and Vulpian. Thus was demonstrated the possibility of increasing or diminishing the virulence of the rabid agent, and of conferring immunity to animals by preventive inoculation.

Another important question was that of knowing if we can, by inoculation, prevent the evolution of hydrophobia when infection has already taken place. Pasteur has decided it in the affirmative. He has attempted to produce immunity in the man who has been bitten by inoculating him with attenuated virus. The French scientist had previously obtained numerous positive results with the dog. In April, 1886, the number of human beings inoculated was 1335. The operation is performed in the following manner: we crush in sterilized bouillon a shred of 2 to 3 centimetres of dried rabid spinal cord (rabbit), and we inject under the skin of the abdomen

$\frac{1}{2}$ to $\frac{3}{4}$ of a cubic centimetre of this preparation. We use at first a very weak virus, on the following days a virus which becomes gradually stronger, and finally a very virulent virus. The number of injections in the beginning was thirteen; later it was reduced. By these inoculations Pasteur has reduced the mortality from 16–80 per cent. to 0.5–1 per cent.

Cantani (of Naples), Ullmann (of Vienna), and Metchnikoff (of Odessa), have expressed themselves favorably upon Pasteur's inoculation. 122 persons have been inoculated by Ullmann; 3 only have died of hydrophobia. Out of 713 which were treated by Metchnikoff, 20 became rabid. Von Frisch (of Vienna), Bordoni-Uffreduzzi (of Turin), de Renzi (of Naples), etc., have contested the prophylactic value of inoculation. Before judging this definitely it is advisable to wait for a greater number of positive facts. At the present time the following arguments may be opposed to it:

1. The exact number of bitten people who are truly *infected* is unknown. According to Bollinger, the proportion varies from 8 to 47 per cent. Pasteur admits as a minimum 16 per cent.

2. Inoculation has never reduced mortality to zero (Metchnikoff has had twenty cases of death).

3. The period of incubation of rabies is too long to enable us to decide definitely at the present time upon the value of inoculation(?).

4. It has not always been established that the people who were inoculated had been bitten by rabid animals; in a certain number of these there was but a suspicion of rabies.

5. Cauterization of the bitten region has almost invariably been made; this fact may also explain the reduction in the death-rate.

6. Pasteur's process is "too premature" in order to become generalized, even by strictly following all the indications which are given by its author.¹

¹ After having recognized that the rabid virulence becomes gradually attenuated by successive evolutions upon the ape, and that it increases later if its virus is brought upon the rabbit, Pasteur succeeded in conferring immunity to the dog by injecting under the skin particles of spinal cord of the rabbit, which increase in virulence. By means of new experiments he established that rabid spinal substance, when subjected to desiccation, loses its virulence gradually and completely after the thirteenth or fourteenth day. He succeeds also in giving immunity to the dog by inoculating it with nervous matter the virulence of which was thus attenuated, or *rarefied virus*. The process had been successful in fifty animals when it was tried upon man. It consisted in making under the skin repeated injections with spinal cord of increasing virulence—from the fourteenth, the thirteenth, etc. . . . and so on to the fifth day.

We agree with Bollinger, who considers as inopportune the creation of institutions for antirabic inoculation. Sanitary prophylaxis, as practised in Germany, and a strict enforcement of the

This treatment, which lasted ten days, was insufficient for serious cases. It was replaced by the so-called *intensive* method. This varies "according to the serious condition, and especially according to the location of the bites. This is our method of procedure: we take a fragment of the spinal cord and crush it in sterilized bouillon and inject a certain dose of this preparation under the skin in the region of the hypochondria. The injection is somewhat painful, but it does not produce any marked accident except in the last two days, when the puncture produces a small erythematous blotch and itching. We begin by using the substance of the spinal cord which is fourteen days old, then the thirteen, and so on progressively until we arrive at the three-days' old virus, the virulence of which is nearly equal to that of the fresh spinal cord. The quantity injected may reach three cubic centimetres for spinal substance which is not very active, those from the fourteenth to the seventh day; we do not exceed two cubic centimetres with spinal substance which is less than seven days old. In serious cases we must act quickly; we start, therefore, with four daily injections. On the first day we inoculate the spinal cord substance of the fourteenth and thirteenth days by means of two punctures, one upon each side of the flank; on the evening of the same day the substances of the twelfth and eleventh. The next day we use those of the tenth to the seven days old. On the third day of treatment we make two injections of the substance which is six days old. Finally we make but one injection daily with the most virulent substances. When reaching the three days' old substance we begin a new series by starting from the five days' old matter, then a third and even a fourth series. In some cases, at the end of the treatment, the patient complains of pain on the surface of the cicatrix; in such cases we start a new inoculation by the intensive method, and we frequently see the symptoms disappear."

It is probable that immunity is produced not by the rabid virus, but by a vaccinal matter which exists in the spinal cord besides the virus, and which is not destroyed by desiccation. In the dog its duration is at least two years.

In human beings the mortality which follows bites made by rabid dogs is nearly 16 per cent. (Leblanc). In France from 1886 to 1889 7919 persons were inoculated, 79 died from hydrophobia (1 per cent.). In 1890 "the treatment was applied to 1546 persons; 5 died during the period of inoculation; 6 were affected by rabies in the fifteen days following the inoculations; 5 only died later. The mortality, taken as a whole, has thus been 0.97 per cent.; if we deduct the cases which died during the treatment, we find 0.71 per cent.; we reach, finally, the figure of 0.32 per cent. by taking into account the persons bitten who died subsequently. (Roger: *Loc. cit.*)

In an interesting communication which was made at the Congress of the Association for the Advancement of Science (Session of Nancy, 1886) du Mesnil called attention to the considerable mortality produced by bites by rabid wolves, and has demonstrated the advantages derived from Pasteur's inoculation: Of 342 persons bitten by rabid wolves 206 died of rabies (60.23 per cent.); of 146 who were bitten and afterward cauterized with nitric acid, caustic potash, ammonia, red iron or thermo-cautery, 87 have died (60.27 per cent.). Out of 19 Russians bitten by a rabid wolf and inoculated in Paris, 3 died (15.70 per cent.) (*Recueil vét.*, 1886). Of 30 animals and 13 men bitten by a rabid wolf, the former (non-inoculated) all died of hydrophobia; one man who was not treated also died rabid; upon the 12 others who underwent the treatment, only one died (mortality 8.33 per cent.).

In 1880 Galtier had already established, by experiments in the sheep and the goat, that immunity against rabies could be conferred to animals by intravenous injections of rabid saliva, and by this method it was possible to prevent, in herbivorous animals

measures prescribed by the law on contagious diseases, the tax on dogs, and the obligatory use of muzzles in large cities, are sufficient to combat hydrophobia. It would, however, be desirable that the scientific side of Pasteur's discovery should be studied and controlled by us, particularly in veterinary schools.

Hœgyes has published a new method of inoculation against rabies. For these inoculations he uses a virus which is more or less concentrated. The spinal substance of the hydrophobic rabbit is treated with salt-water (1 : 1000) and diluted to a variable degree (1 : 5000, 1 : 2000, 1 : 1500, 1 : 200, 1 : 100, 1 : 10). We begin by injecting the weakest emulsions and reach the strongest gradually.

B. Hydrophobia in the Various Domestic Species.

I. RABIES OF THE DOG.

Symptoms. In the dog hydrophobia appears under two clinical forms, which are designated by the expressions of *furious rabies* and *mute rabies*; the first is the more frequent. According to Pasteur, furious rabies occurs when the brain is invaded by the rabid virus, and mute rabies when this reaches the spinal cord first. We may produce the former experimentally by directly depositing the virus upon the surface of the brain, and the latter by injecting it into the subcutaneous connective tissue. Formerly they considered these forms of rabies as two different diseases; but this view should be abandoned.

Hydrophobia is an acute and fatal disease, with typic course.

1. The *furious form of rabies* comprises three symptomatic periods :

- a. A *prodromic or melancholic period* ;
- b. An *irritated or maniacal period* ;
- c. A *paralytic or terminal period*.

and pigs, the development of the disease after bites, even if these existed for some time. The technique of these injections is simple. We need not attempt to avoid contamination of the perivenous tissue; when this slight accident occurs success will nevertheless be insured. By substituting for the saliva an emulsion of virulent nervous matter Nocard and Roux arrested the evolution of rabic infection in sheep which were inoculated in the eye twenty-four hours with virus of street rabies. In the dog rabies is frequently developed after an intravenous injection of rabid virus. However, by repeated injections and by using matter with increasing virulence, we may also confer immunity to this animal (Protopopoff).

Babes and Cerchez have established that the development of inoculated rabies may be prevented or its evolution considerably delayed by the injection of blood of inoculated dogs according to Pasteur's method (Annales de l'Institut Pasteur, 1891).—N. D. T.

a. The duration of the *prodromic period* varies from twelve hours to two days. We observe a change in the habits of the patient: he is depressed, gloomy, or anxious, defiant, restless, cross, and very irritable; he no longer obeys his master, and becomes very capricious. Sometimes he hides in the litter, at other times he often changes his position suddenly; some animals appear exceedingly gentle and affectionate. The region where the virulent bite has been made is sometimes the seat of intense pruritus; the animal licks or bites it; we observe an aberration of taste, showing a certain similarity with the principal manifestations of the *licking-disease* which is seen in the ox. In the beginning the appetite is generally preserved, and some subjects eat with voracity; but they soon refuse food, even that of which they are particularly fond; they lick cold objects or bite anything that is within their reach, and swallow foreign bodies: straw, grass, earth, pebbles, pieces of wood and of glass, rags, blankets, etc., even their excrements; some show symptoms of intense genic excitement, and constantly lick their genital organs or those of other dogs. We sometimes notice a slight dysphagia, abnormal movements of the cervical region, nausea and vomiting. The animals are generally constipated.

b. The *period of irritation* lasts from three to four days. It is essentially characterized by spasmodic rabid attacks, separated by remissions. The paroxysms last for several hours. At the same time, as the restlessness increases, an irresistible force induces the patients to run away. Animals which were always good-natured attempt to break their cage or their chain; pet dogs remain near the door ready to escape. Outside they run around aimlessly; they enter into houses, farms, and wander to far-away localities; they cover enormous distances within a very short time; sometimes they afterward return home. They have then a great propensity to bite. During the paroxysms they lose the instinct of self-preservation. In the beginning the tendency to bite is but slightly marked; the dog *snaps* at empty space as if he wanted to catch flies; he is always very excitable. But soon he becomes aggressive and throws himself blindly upon all objects which he meets: upon animals, especially dogs and cows, the horse, cat, sheep, goat, and poultry; he attacks man, even his master (in general, however, the latter must provoke, menace, or strike him); when locked up he seizes the bars of his cage, and breaks his teeth or fractures his jaws. Some dogs bite the tail, genital organs, or legs; some are

observed tearing pieces of flesh and exposing one or several bones. It is rare that a mad dog avoids people and animals; in exceptional cases, however, he does not attack human beings. At this stage of the affection the alteration of the voice is of great importance for the diagnosis. The rabid dog produces a peculiar hoarse howl, which begins with an indistinct drawn bark and ends in a higher key. This alteration of the voice is no doubt related to paralysis of the vocal cords.

In some subjects we observe symptoms of depression. They are fatigued, dull, and affected by hallucinations; they snap into the air, howl constantly, and are insensible to traumatisms (injuries, blows); their eye is staring and haggard. Dogs which are naturally good natured and well trained may obey their masters up to the last.

c. During the *paralytic period* the animals are emaciated, sometimes unrecognizable; their hair stands erect, and their eyes are sunk in the orbit; the look is threatening and menacing. Finally, we observe various akineses: paralysis of the pharynx (dysphagia and ptyalism), of the lower jaw (permanent opening of the mouth and inertia of the tongue), of the hind quarters (uncertain gait), of the tail, rectum, and bladder. General weakness increases; the paroxysms become more and more rare, and the patients die from the fifth to the eighth day, at the latest on the tenth day. Death is determined by cerebral paralysis or exhaustion. We possess but few exact facts upon the thermic course in rabies. According to Hertwig, the temperature rises 3° C. and sometimes more, only to be rapidly diminished toward the end of the disease. In one case we were able to observe these thermometric variations.

2. *Mute rabies* is distinguished from furious rabies by the absence of the period of irritation. The paralytic symptoms, especially paralysis of the lower jaw, appear much more rapidly than in the other form. Quite frequently death occurs within from two to three days.

Some authors have described an abortive form of rabies; they have seen the disease arrested in its first stage, retrocede, and terminate by cure. But these facts which they have mentioned do not seem to us convincing.

The public generally have a very erroneous idea concerning the symptoms of rabies in the dog. They believe that the rabid animal runs straight ahead, with his tail between his hind legs; that he is

affected by intense hydrophobia, with injected eyes and foaming mouth, etc. Rabid dogs are not at all hydrophobic; they are often seen to lap water; some will even swim rivers.

The symptoms of *rabies in man* differ considerably from those which we have just described in the dog. However, we may also recognize three periods. During the prodromic phase we observe general uneasiness, pain on the surface of the bitten region, a tumefaction of the corresponding ganglions, and apathy for liquids. At the second period, which is called *hydrophobic*, we notice reflex spasms, delirium, and hallucinations. The spasms are observed in the pharynx and œsophagus; they are produced by the sight of water (hydrophobia), when the patient undergoes the sensation of thirst if we speak to him of drinking, or if he thinks of it; we observe also oppression, respiratory spasms, convulsions, and great terror. The thirst is intense; dysphagia occasions ptyalism. The temperature rises; in some cases hyperthermia is very pronounced. The terminal period is characterized by paralytic states and spasmodic attacks. Death happens within from two to four days. Authors have mentioned a few examples of recovery.

Besides prophylactic cauterization the treatment consists of general anesthesia by chloroform, chloral hydrate, bromide of potassium. We might also try curare.

Diagnosis and differential diagnosis. The diagnosis of rabies of the dog is cleared up by the antecedents of the animal, by the anamnesis, especially by the information that the dog has been bitten. Among the clinical symptoms the most important are modifications of character, alteration of the voice, tendency of the animal to escape, propensity to bite, paralytic phenomena, and the typic course of the affection. The information obtained by the autopsy permits us almost always to express ourselves with certainty. The diagnosis of mute rabies is sometimes difficult, especially for dogs which are well kept, and in cases where the history is erroneous or incomplete. In such cases hydrophobia may only be affirmed by a test inoculation. It is preferable to make an intracranial injection (dog or rabbit) with fresh nervous matter, which should be obtained from the spine or the brain. Inoculation is indicated in every case where man has been bitten.

The diseases which may be confounded with rabies are numerous: cerebral hyperemia, encephalitis, parasites and tumors of the brain (*Tenia echinococcus*), pyloric or intestinal obstruction, perforation

of the stomach (fish-bone), parasites of the nasal cavities (*Pen-tastoma tænioides*), pharyngitis, a bone fast between two teeth (Johne), a foreign body arrested in the œsophagus, paralysis of the jaw (Münich), facial paralysis, hemiplegia (Sewell), troubles which are produced in the female dog by depriving her of her pups (Colin), sunstroke (Fünfstück), habit of biting, viciousness, etc. In doubtful cases intracranial inoculation fixes the diagnosis.

II. RABIES OF THE OX.

Etiology. After the dog, rabies is more frequently seen in the ox than in any other domestic animal. This frequency seems to depend upon the predominance of subjects of the bovine species and to the aversion of the dog toward the ox. In this latter the disease is generally inoculated by the bite of a rabid dog; more rarely it is directly transmitted from ruminant to ruminant (Cope and Horsley have mentioned this mode of contamination in stags in Richmond Park, England); sometimes it comes from the horse, cat, or pig. The bites are generally located upon the face, lips, or posterior members. The period of inoculation is from four to eight weeks on the average; in some cases it lasts for several months. It is prolonged in exceptional cases to 203 days (Gielen), two years, and four months (Morro). The shortest duration is one week. According to Spinola, the state of gestation increases the period of incubation in the cow. We know of two cases of intra-placental transmission of the infection.

Symptoms.¹ In the ox rabies is generally marked by stamp-

¹ Ladague has published in the *Receuil* of 1885 a description of hydrophobia of the ox. He observed twenty-seven cases in a herd of eighty animals, most of which had been bitten by the watch-dog. He details thus the principal symptoms of the disease:

"On the first day: Slight colics, or, at all events, something like it, for the animal rises and lies down frequently, exaltation of the senses, abrupt and much marked rise of temperature (40°), pruriginous pain in the bitten region.

"On the second day: Less agitation, slight tenesmus, lessening of the heat in the region where the bite has been made, lowering of the temperature.

"On the third day: Beginning paraplegia, marked tenesmus with passage of fecal matter covered with yellowish-brown mucus, lessened reflex of the spinal column, decreasing temperature which only ceases with death, bellowing.

"On the fourth day: Complete paraplegia, violent tenesmus, frothy mucus upon the fauces, thread-like clear saliva, more frequent bellowing.

"On the fifth day: Numerous symptoms of tenesmus, abundant foamy mucus ejected by the anus, considerable lowering of temperature, frothy saliva, and rare bellowing.

"I have not observed any depravity of the taste during these five days, notwith-

ing and by butting, which is so violent that the horns may be fractured. There are animals which scratch the ground continually with their feet or dig it up with their horns; the tendency to bite is more rare. Their expression is wild, the eye prominent, and the conjunctiva red. Some subjects bellow continually; here, also, we notice a peculiar modification of the voice. Abundant salivation, constipation, and permanent ineffectual expulsive efforts are constant manifestations of rabies in the ox; rectal tenesmus is sometimes the first symptom of it. Some patients gape always. In some instances the genesic instinct is much excited. At a more advanced period the gait becomes stiff and the hind-quarters paralyzed. Emaciation is rapidly marked. Death occurs within four or five days.

Differential diagnosis. We must especially differentiate from rabies: encephalitis, tubercular bacillary meningitis, poisonings, and more particularly lead-poisoning, gastro-enteritis (Papa), foreign bodies in the œsophagus, tumors of the plexus (Meyer), dermanyssees which are located in the external auditory canal (Stadler, Schuemacher), malignant catarrhal fever, anthrax, bovine plague, sun-stroke, and phenomena of excitement occasioned by the abrupt sequestration of animals which are used to living in freedom (Köhne).

III. RABIES OF THE HORSE.

Etiology. In the horse rabies is usually consecutive to bites made by the dog, more rarely by rabid wolves or foxes. The period of incubation averages from four to eight weeks; its maximal duration, according to most authors, is twelve weeks. It may, however, be longer in exceptional cases; in one instance it was two hundred and eighty-three days (Roll), and in another twenty months (Gotteswinter).

Symptoms. We observe at first modifications of the disposition and character; the animal is anxious and nervous; he scratches with the foot, and neighs, bites the manger, the sides of his stall,

standing that I arranged to have various foreign matters placed within the reach of the animals."

Ladague saw two remarkable cases of *intermittence* upon these rabid animals. An eighteen months old bull, after having shown all the symptoms of rabies for three days, recovered, and for five weeks his condition appeared to be normal, then the disease returned anew and ended in death within four days. In a heifer which died twenty-four hours after this bull they observed at an interval of one month two attacks of similar phenomena.—N. D. T.

and the objects placed within his reach. We may observe fractures of the incisors and of the jaws; the region where the bite has been made—ordinarily the lips, nose, or a front leg—is the seat of intense pruritus. Some horses, actuated by an irresistible impulse, attack man with their teeth and their feet; others bite, lacerate themselves, and tear out shreds of skin and muscles, testicles, etc.; others, also, masticate anything which they may grasp with their teeth, they seize sticks that are held before them, eat and swallow manure; some patients have the physiognomy of the immobile horse. In the beginning they ingest food with avidity; later, inappetence becomes absolute; thirst is always very great. We observe also a more or less marked genic excitement. The stallions have erections and attempt to perform the service; there is also incontinence of sperm. The over-excited mares neigh loudly and make violent efforts of micturition; the vulva is the seat of permanent pruritus. When the pharynx is paralyzed the food is ejected by the nose, the jaws are continually moving and violently closed at certain times; they grind their teeth, the head and tail are constantly agitated. Besides we see convulsions, muscular spasms in various regions: upon the head (lips, cheeks), neck, upon the pectoral and abdominal walls, and symptoms of colics (groans, decubitus, expulsive efforts, rectal prolapsus). Finally, general paralysis follows. The walk is uncertain, the fetlocks bend, the patients stagger; soon we observe the symptoms of paraplegia. In some cases the paralytic phenomena begin in the region where the rabid virus has been deposited [(lips, front legs) Gerlach, Ménézin]. The temperature, which is normal in the beginning, rises rapidly to 40° C. and above; the number of pulsations is increased, it may be doubled or trebled. When the end is near we observe sweating spells. Death, which is “apoplectiform,” sometimes occurs within twenty-four hours; but the average duration of the disease is from four to six days. In the horse mute rabies is more frequent than in the ox.

Differential diagnosis. Rabies of solipedes may be confounded with encephalitis, the paroxysms which occur during the course of immobility, cerebral abscess (Kopp), violent *heats*, colics, gastritis (Olivero and Allunanno), laceration of the stomach, and paraplegia.

In order to establish a diagnosis we must especially consider psychic troubles, the tendency to bite, efforts of defecation and micturition, colics, local pruritus, and an exaltation of the genic instinct.

IV. RABIES OF THE CAT.

Symptoms. After a period of incubation of two to four weeks on an average, rabies appears. The patients try to run away; they become aggressive, bite and scratch. The voice is altered and hoarse. Of all rabid animals the cat is the most dangerous to man; it jumps at the face and attempts to tear it with the teeth and claws. It no longer fears the dog, which it attacks boldly.

Death occurs from the second to the fourth day. In the cat we may confound rabies with nervous accidents produced by helminthiasis.

V. RABIES OF THE PIG.

Symptoms. The rabid pig grunts, howls, and performs uncontrollable movements, or hides in its litter; it is restless, over-excited in some instances; it attempts to bite, attacks man, animals, and frequently ingests indigestible foreign bodies; a foamy saliva runs out of the mouth. In the region where the bite was made we frequently observe at the time when the rabies appears a marked inflammation and violent pruritus. The duration of the disease is very short; it seldom exceeds twenty-four or forty-eight hours. The period of incubation averages from two to three weeks. According to statistics, it varies between six days and one hundred and seventy-nine days. Mute rabies has not yet been reported in the pig.

VI. RABIES OF SHEEP AND GOAT.

Symptoms. The rabid sheep shows great excitement; we observe intense pruritus upon the surface of the bites; it licks these regions and strips them of their wool. The genic sense is considerably increased; we hear a hoarse and loud bleating. The animal fears man no longer; when approached it becomes aggressive, it stamps, and bites sticks when they are presented to it; it also bites all objects which it may reach—the manger, rack, etc.; it rears against the walls of the sheepfold and emits a wild respiratory bruit; the eye is haggard, and the expression stupefied. Weakness supervenes rapidly. Death occurs within three or four days. The period of incubation is from three to four weeks.

In most cases the sheep is contaminated by the dog; as a rule,

a large number of animals are bitten; we have frequently seen nearly a whole flock die. Some instances have been mentioned of transmission of rabies to the sheep by the wolf and the bull (Bourrel).

In the goat the symptoms are nearly the same as in the sheep.

VII. RABIES OF POULTRY.

Symptoms. Rabid birds are nervous, anxious, and over-excited; they run in all directions, make unusual jumps, utter cries, attack other birds with bill and claws; sometimes they throw themselves upon man, tear his clothing, and attempt to swallow pieces of rags; their voice is altered, and becomes hoarse; finally, we see the appearance of paralytic symptoms. The patients die within two or three days. The period of incubation may vary from six weeks to eleven months.

APHTHOUS FEVER.

Foot and Mouth Disease.

HISTORY. This contagious disease has been known for a long time. It is also designated by the names *aphthous disease*, *vesicular disease*, *mouth rot*, *mouth disease*, *cocotte*, and *epizootic aphthæ*. Toward the middle of the eighteenth century, at the period when it occasioned great losses in Germany and in England, several authors gave a good description of it. From 1809 to 1812, and from 1819 to 1823, it existed in southern Germany, in Switzerland, and in Italy (Buniva, Handel, Lux, and others). In 1839 it was introduced into England, where, since that date, we have seen ten great epizootics of it (Brown). From 1840 to 1860 it extended throughout all Europe; it existed with marked intensity during the years 1845-46, 1855-57, 1862, 1869, 1871-74, 1875-77, 1883-84.

In 1871 it affected 700,000 animals in France, and nearly the same number in England; 14,000 died (1 per cent.). In Würtemberg, in 1872, it affected 50,000 animals, 1500 of which died (3 per cent.). At the same period, in the Duchy of Baden, it was observed in 150,000 animals. In 1883 they counted nearly a half-million of aphthous fever subjects in Great Britain, 60,000 in Prussia, as many in Austria and Italy, and more than 100,000 in Bavaria (83,000 bovines, 15,000 sheep, 5000 pigs). In 1886, in

the German Empire, aphthous fever was seen in 13,603 animals (5366 oxen, 6388 sheep, 1839 pigs, 10 goats). It existed particularly in the district of Königsberg, in Lower Alsace, and southern Bavaria. In Austria the number of patients was 1503 in 1886; in Switzerland, 2710 in 1887; in Prussia, 59,708 in 1884, and 12,068 in 1885; in Bavaria, 105,100 in 1884, and 6842 in 1885.¹

Etiology. Aphthous fever is an acute infectious disease belonging to the group of exanthemas, the germ of which is *volatile* and fixed, and may be transmitted directly or indirectly to healthy animals. The specific agent is contained in the liquid of the vesicles and ulcerations, in saliva, milk, feces, urine, in the expired air, and the products of perspiration. In the pure liquid of the vesicle Nosotti has found a constant micrococcus which easily takes the aniline colors; he has cultivated and inoculated it with success. According to Klein, the micrococci of aphthous fever appear in the shape of diplococci or streptococci, and sometimes form little chains with a score of sections; if grown in certain mediums (coagulated serum, gelatin), they produce characteristic cultures—thin membranes, which are marked with fine points formed by very delicate globules. By mixing these cultures with food Klein has produced aphthous fever in sheep; subcutaneous inoculations have remained without effect.

The contagious germ possesses great resistance to the causes of destruction; in infected stables and manure it may preserve its activity for months, even a whole year. We may thus understand how cured animals sometimes remain contaminated for a very long time. According to Schleg, the infectious element of milk reaches its height at the febrile period of the disease; later it diminishes; it is destroyed by boiling.

The propagation of aphthous epizootics occurs especially by ways of transit. We have frequently observed their extension from east to west, and their progression with "lightning rapidity" from the Caspian Sea to the Atlantic. In general, the rapidity of their expansion in a country is in proportion to the extension of railroads. The principal factors of mediate contagion are wagons, innkeepers' stables, markets, pastures, and common troughs, dealers,

¹ In France, during the year 1887, aphthous fever appeared in twenty-seven departments, but it was of a mild character. It affected 471 animals of the bovine species, 1011 sheep and 41 pigs. Of these 1523 patients, 73 died (4.8 per cent.)—19 bovines, 48 sheep, and 6 pigs—representing a value of \$1333. Tisserand: Rapport sur le Service des Epizootics en 1887.—N. D. T.

veterinarians, and persons who have charge of animals, flocks of sheep, bulls, feed, litter, and manure. Herds of pigs in transit have been shown to be particularly dangerous. In the same stable transmission may also occur directly from sick to healthy animals (licking), or by persons who attend to milking (hands), and by inspired air. Young sucklings become infected by the ingestion of milk.

Nothing positive is known concerning the mode of entrance of the pathogenic agent. It is probable that it penetrates into the system quite as easily by the digestive apparatus as by the lungs.

The patients of one species of animals may contaminate subjects of other susceptible species (ox, sheep, pig).

AFFECTED ANIMALS. Aphthous fever is a disease of bisulcates. It is most commonly observed in the ox, sheep, pig, and goat; but the horse, dog, cat, and poultry are not exempt from it. Man himself contracts it quite frequently. The wild bisulcates (stag, deer, chamois) and ruminants which are kept in zoological gardens (camels, llama, giraffe, antelope, buffalo, American bison, etc.) are also liable to contract this affection. According to Peschel, draught oxen are less exposed to it than other animals of the same species. A first infection does not confer immunity; the disease may return several times during the course of a year.

Aphthous fever is of great importance from an economical standpoint, especially on account of its rapid extension over vast territories. It may affect 25 to 50 per cent. of the bovine species; it requires a suppression of commerce and general trade; it also considerably diminishes the mammary secretion, prevents the use of draught animals, and always lead to more or less marked emaciation. In England, in 1883, the losses occasioned by aphthous fever were estimated at \$5,000,000, and in France for the year 1871 at \$7,000,000. In Switzerland the loss is nearly \$2,000,000.

Symptoms in the Ox. In this species aphthous fever is characterized by vesicles and ulcerations which appear on the buccal mucous membrane (aphthous stomatitis), on the skin of the coronary and of the inter-digital integument (disease of the hoof).

In the sheep, goat, and pig we generally find only the disease of the hoof.

1. **APHTHOUS STOMATITIS.** After an average period of incubation of from three to five days (from twenty-four to forty-eight hours only in some cases) the disease manifests itself by a fever of infection of medium intensity (up to 40° C.), which persists up to

the time of the eruption. The buccal mucous membrane is red, the appetite and lacteal secretions are diminished; ordinarily the salivation is increased. Within two or three days whitish-gray vesicles of the size of a hemp-seed appear upon the gums, on the edges and lower face of the tongue, on the mucous membrane of the cheeks, and on the lips; they gradually become enlarged; many reach or exceed the dimensions of a silver dollar; sometimes several are confluent; they are always sharply defined in the depth of the mucous membrane. In the beginning they contain a limpid, yellowish liquid, which later becomes turbid. On being torn they produce red wounds, which are very sensitive and deprived of epithelium; these are the aphthous erosions. They sometimes become slowly covered by an epithelial layer; at other times they are transformed into ulcerations having a pale, bleached fundus or bottom, and which cicatrize only after a long period. At this stage we observe ptyalism: the saliva falls out of the mouth in long threads and accumulates upon the ground or in the manger. The exanthema extends quickly to the muzzle. The patients lose flesh rapidly. The milk is altered: it has a yellowish-white coloration and a mucous consistency similar to that of colostrum, and a very disagreeable taste; it is difficult to make butter or cheese with it.

• This clinical tableau, the manifestations of which evolve in from eight to fourteen days, may be modified by various complications. In the cow the exanthema is frequently communicated to the udder and teats, where the infectious agent may be carried by the person who practises the milking. From the galactophorous canals the inflammatory process may be propagated to the parenchyma of the gland (parenchymatous mammitis). The aphthæ which develop upon the pharyngeal mucous membrane determine symptoms of angina: salivation, regurgitation, dysphagia, and cough; in that case, when drinks are administered, it is not rare to produce a pneumonia by foreign bodies. As in other acute exanthema, we often observe the symptoms of a catarrhal phlegmasia of the nasal and bronchial mucous membrane. When the process is very intense upon the buccal mucous membrane we may notice an exudate similar to that of croup; the desquamated epithelium becomes decomposed and gives off a fetid odor (mouth rot). In sucking calves and even in adult animals we also observe the symptoms of a serious gastro-intestinal disease.

When the matrix of the horns becomes inflamed, these appendages become detached by slight mechanical action, and vesicles often appear upon the skin of the neighboring regions. We may also find aphthæ upon the vaginal mucous membrane and the vulva, upon the skin (especially upon the abdomen and the chest), even upon the cornea. The females abort frequently. In some cases the exanthema is entirely wanting; then, at times, the disease offers a certain analogy with anthrax fever. It is possible that aphthous fever may complicate pleuro-pneumonia, but only when the febrile period of the latter is over, never at the pyretic stage (Prietsch). In some instances we see cases of *apoplectiform* death, the causes of which are sometimes a multiple embolic myocarditis (Johns), at other times asphyxia produced by shreds of epithelium introduced into the respiratory passages (Lydtin).

TRANSMISSION OF APHTHOUS FEVER TO MAN is not rare. The veterinarian has oftener occasion to observe it than the physician. The use of milk from aphthous cows contaminates children quite frequently, and is fatal to them. This may also happen through ingestion of butter or cheese made of milk coming from aphthous animals or also directly through wounds of the arms, hands, or by intermediary agents. In man the symptoms are: fever, digestive troubles, vesicular eruption upon the lips, the buccal and pharyngeal mucous membranes (angina). The disease does not seem to be transmissible through the meat of diseased animals. Perhaps the serious affections of the skin which were observed to develop in children after vaccination (especially in 1883-84) may have been determined by mistaking the mammary eruption of aphthous fever for cowpox.

2. SYMPTOMS OF APHTHOUS FEVER ON THE HOOF. The symptoms of the foot lesions generally follow those of aphthous stomatitis; they may also appear simultaneously. The skin of the coronet, especially in the interdigital space and in the posterior region thereof, is red, hot, painful, and tumefied. These phenomena are observed in one foot, in several, or upon all four, at the same time. Within from twenty-four to forty-eight hours, the inflamed integument becomes covered with small vesicles, varying in size from a pea to a hazelnut, and containing a yellowish limpid liquid in the beginning, but later this becomes turbid and viscous; these phlyctenes soon appear. The walk is stiff, the animals are lame; in the stable they remain almost constantly down. If the

process evolves regularly the wounds consecutive to rupture of the vesicles form cicatrices within eight to fourteen days.

Grave complications are quite common. If the affection is neglected and the patients are left upon damp, unclean ground, soiled with the discharges, or if they are compelled to walk upon pebbled roads, we soon see upon the integument of the foot intense inflammatory, erysipelatous, septic processes and ulcerations. Then follow abscesses, whitlow under all forms, suppurating arthritis, necrosis of the bone, falling out of the hoof, and, lastly, pyemia. The prominent regions of the body show patches of sloughing skin produced by the prolonged decubitus.

Differential diagnosis. Aphthous fever may be confounded with cauterizations and burns of the buccal membrane, also with mycotic stomatitis produced by the fungi of colza [(*Polydesmus exitiosus*) (Brümmer)], mercurial stomatitis, poisonings by potato leaves, whitlow, simple inflammation of the interdigital space, malt eczema, simple eczema (in one case of this latter trouble, Heminger has observed infectious lesions of the buccal mucous membrane which were produced by licking), also with anthrax, variola, and bovine plague. The cases described under the name of "*sporadic aphthous fever*" are no doubt determined by traumatic actions.

Symptoms in the sheep. In the sheep and goat aphthous fever is usually located on the toes. The inflammatory process evolves upon the coronet, especially on its posterior portion toward the plantar cushion; the region of the coronet is tumefied, red, and exudes a lymphoid liquid. The patients limp and drag behind the flock. We sometimes observe an exanthematous eruption upon the pad of the upper jaw, upon the vulva and the vaginal mucous membrane. In sucking lambs the disease is particularly serious; death often occurs within a very short time, and is preceded by marked gastric symptoms. In sheep aphthous fever has a slower course than in the ox and the pig.

Among its complications we must mention a *malignant disease of the hoof*. We therefore call a mixed disease an aphthous inflammation complicated by whitlow (infectious inflammation of the extremities which is due to the action of the septic products coming from the soil). The lower part of the legs is much tumefied and red; we observe lymphangitis and phlegmons, putrid decomposition of the secretions, suppuration under the hoof, then detachment of the coronary band, falling of the nails, and suppurative inflamma-

tion of the interdigital gland, purulent arthritis, bony necrosis, and pyemia. The indirect causes of these complications are dampness and uncleanness of the stables, long walks, excessive wear of the hoof, and wounds produced by stubbles. This disease has been confounded with petechia, a simple traumatic eczema of the interdigital integument having no contagious character.

Infectious petechia is nothing but aphthous fever itself. Spinola has insisted on the fact that *malignant aphthous fever*, of which we have just spoken, is a complication of the disease of the foot (simple aphthous fever). We have ourselves had the opportunity of verifying this opinion. According to Holzendorff, we sometimes find fresh aphthæ upon one foot, while the others show all the symptoms of *malignant disease of the hoof*. In the ox we observe the same complications; but in this latter species the patients are usually killed before such serious lesions have had time to be produced. Siedamgrotzky has demonstrated experimentally that this malignant disease of the foot is contagious. By comparing *malignant disease of the foot* and "aphthous disease of the foot," and considering them from the standpoint of their symptomatology, course, and period of incubation, we recognize that Siedamgrotzky was mistaken in devoting to the former (*Spanish or French hoof disease*) a special chapter in the new edition of the *Agricultural Veterinary Medicine* of Haubner.

APHTHOUS FEVER OF THE PIG. In this animal also the disease appears at first in the feet, more rarely in the buccal mucous membrane. Its manifestations are the same as those of the sheep. Aphthæ, inflammation of the coronet, and lameness (*lame disease*), are the principal symptoms. We observe sometimes complications which characterize *malignant disease of the hoof*. They occur especially on individuals of herds which have to make long journeys. The aphthæ may also appear upon the mucous membrane of the groin, when the vesicles may be of the size of a pigeon's egg to that of a nut. Young suckling pigs are particularly sensitive to aphthous fever. It may be confounded with contusions of the feet, resulting from long journeys.

APHTHOUS FEVER OF THE HORSE. In the horse we observe only aphthous stomatitis. This animal is contaminated by the ox, the goat, sheep, and pig. Bräuer has seen a horse which contracted the disease by licking an aphthous ox. There are cases where infection takes place by the intermediation of buckets. In a large

number of observations of "aphthous fever of the horse" we no doubt have to deal with contagious or catarrhal pustular stomatitis. But the existence of aphthous fever in the horse is demonstrated in an undeniable manner. The symptoms are the same as in the ox: fever, inappetence, typic eruption upon the buccal mucous membrane, upon the lips, the nasal mucous membrane, and the skin, gastro-intestinal and bronchial catarrh. Croupous inflammations are more frequent in the horse than in the ox.

APHTHOUS FEVER OF THE DOG. This is very rare. However, in dogs kept in infected stables or in contact with subjects affected with aphthous fever we have observed aphthous and later ulcerative stomatitis, accompanied by phlegmasia of the skin of the interdigital spaces and the plantar cushions of the sole.

APHTHOUS FEVER OF THE CAT. In the cat the disease is marked by fever, vomiting, lameness, by aphthæ and ulcerations of the mouth, tongue, the upper lip, and the face.

APHTHOUS FEVER OF POULTRY. It is very rare, and is characterized by vesicles upon the buccal, pharyngeal, laryngeal, and nasal mucous membrane, also upon the conjunctiva, the comb, the feet, and the interdigital membranes. Kitt has not succeeded in transmitting the affection experimentally to poultry.

Course and prognosis. The course of aphthous fever varies with the periods. Sometimes the epizootics are relatively mild, at other times they are serious. In general, the infectious process has a typic evolution; recovery occurs in nearly all cases within from two to three weeks; the mortality is frequently *nil*. Sometimes the mortality reaches 1 per cent. Some malignant epizootics have killed 5 per cent. of adult animals, and 50 to 80 per cent. of sucklings; most of these latter perished a few days after birth. Animals which are badly fed and weak are less resistant than those in good condition.

The duration of the epizootic in a stable or a flock is from four to six weeks; its extension is more or less rapid according to the period. As soon as a cure is obtained the animals gain flesh rapidly. But the disease may leave persisting emaciation, diminution of milk secretion, broken-wind, mammitis, chronic diseases of the hoof, pruriginous cutaneous eruptions leading to dropping of the hair, and persistent lameness, etc. Besides, reproduction is hindered; most newborn animals die.

Pathological anatomy. At the autopsy we invariably observe,

as the cause of death, either intense local alterations or visceral complication. We may observe pneumonia by foreign bodies, specific vesicular or ulcerous gastro-enteritis (Engesser, John), aphthæ in the larynx and the pharynx, myocarditis, parenchymatous alterations of the liver, spleen, kidneys, etc. We may also find lesions of acute cerebral dropsy.

Treatment. The prophylaxis requires isolation of the patients and use of the milk only after it has been submitted to prolonged boiling. When the exanthema follows a mild and regular course it is mostly sufficient to begin a dietetic regimen. We give to the animals flour and bran slops and plenty of clean water. The litter must be often removed, and the soil kept dry with tan bark, plaster, or *kainite* (chemical fattening substance containing potash) (sulphate of potassium). We may resort to therapeutic agents only in serious cases. Ulcerations of the mouth should be treated by astringent and disinfecting lotions (vinegar and sea salt, alum, borax, a decoction of tannin, nitrate of silver); the disease of the hoof by phenicated water, cresolated water, a solution of sulphate of copper, tar, etc.; and mammitis with camphor ointment, salicylated or boric glycerin. (Concerning measures of sanitary police, see the law upon contagious diseases.)

Inoculation. In the beginning of this century Buniva advised inoculation of apthous fever with the object of lessening the duration of epizootics. Since his time this operation has been done many times (Ercolani, Brauell, Reuner, Hoffmann, Wirth, Spinola, Hertwig, Lewes, Brandes, etc.). It deserves to be recommended; it not only shortens the duration of the disease, but apthous fever which is transmitted in this manner generally follows a regular course and remains localized upon the buccal membrane. The *modus operandi* of the inoculation is very simple. The buccal membrane of all the animals of the infected stable must be rubbed or scarified, then covered with virulent saliva, or the infecting matter should be deposited by means of the lancet upon any region of the skin, or we may pass setons saturated with the infecting matter under the integument of the ears or of the tail. In the pig the inoculation is made upon the groin. Within twenty-four hours the fever appears; on the third day we observe aphthæ, which rupture three days later. From the tenth day the process of cicatrization begins. Inoculated apthous fever is almost always mild. But if the *inoculation of necessity* is of

advantage, the same is not the case with *preventive inoculation*; aphthous fever is a relapsing contagious disease. This fact explains the inefficiency of inoculation of pure virus by subcutaneous injection made in Italy in 1883 (Nosatti).

VARIOLA: VARIOLOID. ~

A. Generalities of Variola.

HISTORY. In the lower animals, as in man, variola has been known for many centuries. Sheep-rot or scab, with the exception of human variola, is perhaps the only independent form in the group of varioloid diseases. This disease also appears to come from countries of the Orient (the march of epidemics is from east to west). It was first mentioned in England in 1275. In France it was described for the first time by Rabelais in 1578; inoculation for this disease is also of very ancient date. In 1691 scab was noticed in Italy by Ramazzini, and in 1698 in Germany by Stegman. Its contagiousness was studied by Bomgelat in 1763. Toward the end of the eighteenth century (in Germany in 1770) preventive inoculation was practised on a large scale. The generalization of this operation, together with the introduction of Spanish sheep into the country for the purpose of improving the native breeds, resulted in considerably extending this disease. In France alone, in 1819, it caused a loss of more than one million of sheep, and in Austria, in 1823, more than half a million. In Germany this variola of sheep is said to have almost entirely disappeared through the enforcement of severe sanitary police measures. In Bavaria, in Würtemberg, and in Baden it has not been observed for several years. At the present time it is still to be found in eastern Prussia, in Russia, in Hungary, in France, and in England, and also in many other countries.

For several centuries variola of the cow (cowpox) has been known in England. In 1796 Jenner transmitted it to man, and demonstrated that this operation conferred immunity to human variola. Since this period vaccination has become rapidly generalized.¹

¹ Cowpox is not the variola of the cow, but is the horsepox, phlyctenular herpes, pemphigoid rhinitis, coital exanthema, contagious stomatitis, mal des talons or grease of Jenner, etc., variola of the horse accidentally communicated to the cow. Variola proper of the bovine species is still unknown.—L. T.

Variola of the horse (horsepox) seems to have been much more common formerly than it is at the present time. Jenner and Sacco frequently observed it at the beginning of this century; the former considered it as the original disease from which was developed cowpox. According to Röhl, it spread very extensively in Vienna, in 1855, among young cavalry horses. As has been stated by H. Bouley, cowpox is very often seen in Paris and its environs. In Germany no case of horsepox has been observed for "several decades."² In 1838 Hertwig inoculated himself and several other persons with variola of the horse; an eruption which resembled that of cowpox covered the hands and arms. Previously we had observed a number of cases of the transmission of equine variola to man, especially upon horseshoers through the manipulations required in shoeing; we have also noticed that this benign infection conferred immunity from human variola.

THE VARIOLA OF MAN was known in the earliest antiquity. In the Middle Ages devastating epidemics spread over western Europe; in the last century more than a half-million people perished annually from this disease. Varioloid inoculation is a very ancient operation. It appears to have had its origin in India or China; afterward it was brought into Asia Minor and Constantinople, when Lady Wortley Montague caused her son to be variolized in 1717. Then the practice of inoculation passed into England. In 1798 Jenner discovered vaccination, which replaced variolization.

AFFECTED ANIMALS. The varioloid diseases are observed for the most part in domestic animals (sheep, ox, horse, goat, pig, dog) and upon man; they are also noticed in the monkey, the chamois, and the buffalo. But in all these species they do not seem to constitute primary and independent diseases. Turner, Leroi, Ceely, Sunderland, and Thiele have made reports to show the relation between human variola and cowpox. More recently Bollinger and Roloff have asserted that cowpox was developed from the variola of man. According to Bollinger, there exist but two principal variolas: the *variola of man* and *that of the sheep* (clavelée, scab). All the others (horsepox, cowpox, variola of pigs, of goats, and the dog) are nothing but erratic varieties. The intimate relationship which exists between these different affections is sufficiently demonstrated by their transmissibility from one animal species to another, and by the immunity conferred by one against all the

¹ See Coital Exanthema and Contagious Pustular Stomatitis.

others. The identity of animal and human variola is established by facts of the same order.¹

Etiology. The variolas are contagious febrile diseases indicated by an acute infectious exanthema with a typical course. The contagion is fixed and *volatile*; it exists in the contents of the pustules, the scabs, the blood, the secretions, the excretions, the expired air, and the products of cutaneous perspiration. It can spread over considerable distance (200 metres and more for clavelée—scab). The contamination generally takes place through the respiratory tracts; it can also be produced by the skin, and then we observe a localized eruption (ox). Chauveau has demonstrated that the virulence is inherent in the micro-organisms, and not in the liquid part of the variolic secretion. The contagious element itself has not been recognized, but it is surely of a microbic nature. A number of bacteriologists (Zürn, Haller, Cohn, Weigert, Klein, Toussaint, Semmer, Ranpach, Plant, Guttmann, Pfeiffer, and others) have discovered micro-organisms in the variolic liquids. Guttmann has found a micrococcus (*Staphylococcus cereus albus variolæ*) which he cultivated. Pfeiffer has described spheroidal corpuscles—also noticed by other observers; these corpuscles are formed of a homo-

¹ The experiments made in 1863, 1864, and 1865 by the Lyons Commission, which was inspired by Professor Chauveau, have demonstrated that variola (smallpox) and vaccinia (cowpox) are two specifically distinct diseases, and that the simple passage of variola virus through the organism of the ox or horse is incapable of transforming this varioline virus into vaccine virus. According to Ceely, other experimenters have in their own imagination obtained this transformation. Haccius and Eternod have again declared that they obtained this result by cultivating the varioline virus upon a calf. The experiments made by Chauveau at Alfort have demonstrated the error made by these authors, and re-established the autonomy of vaccine, and also confirmed the results obtained more than twenty years ago by the Lyons Commission. In the works where these experiments are reported the eminent physiologist compares the effects of the varioline vaccine and charbon virus, and points out the radical difference which exists between the *attenuation* and *transformation* of virus. Here are the conclusions:

"1. That vaccinal virus never gives variola of man.

"2. The varioline virus never gives vaccine to the ox or the horse.

"3. That vaccine is not the attenuated varioline, and is not to be compared to the benign charbon infection communicated to animals by inoculation with attenuated charbon virus.

"4. If the vaccine virus is derived from the varioline, it is by reason of a *radical transformation* of the varioline virus.

"5. To conclude, these latter propositions carry another and more general one, which is this:

"The attenuation of virus is not an operation which we are able to identify with their transformation. In the actual state of things the distinction between this and that is necessarily improved." (Chauveau: Bull. de l'Acad. de Méd., 1891.)—N. D. T.

geneous capsule and a finely granular nucleus; their dimensions vary between $20\ \mu$ and $30\ \mu$; their capsule sometimes encloses several protoplasmic corpuscles; they produce spores (Sporozoïd), but up to the present time there is nothing upon which to establish the pathogenic elements of these organisms. In animals, in the contents of the variola pustules, Van der Loeff discovered mobile corpuscles (Rhizopods, apparently of the family of Protozoa), which he considered as the active elements of the virulent process.

The contagious principle of variola has a great resistance to all destructive influences; kept from putrefaction, it may be preserved for months (in the stables it retains its activity from four to six weeks). It is destroyed by putrefaction, by high temperature, and by carbolic acid, etc. A first attack confers immunity. In the sheep this persists during its whole life.

Symptoms. Custom divides the clinical appearances of variola into five periods, permitting us to point out the typical progress of the exanthema.

1. The *period of incubation*. This is the lapse of time which supervenes between the moment the infectious agent enters the organism and the appearance of the first symptoms. The average duration is about one week.

2. The *initial period (stadium prodromorum)*. This is indicated by the fever, a catarrhal affection of the mucous membrane, by the erythema, etc. This duration is from one to two days.

3. The *period of eruption*. This is announced by the appearance upon the skin of red spots resembling the bites of fleas, which become transformed into small pimples of the same color, hard, of the size of a pin-head, and surrounded by a hyperemic zone. They become developed, preferably on the surface of cutaneous follicles; within a few days their central and prominent part becomes pale; they are transformed into white vesicles, in the centre of which we notice a depression (umbilicated); then their contents are limpid and serous. The variola has reached maturity; it is the most favorable moment for inoculation. The whole phase lasts from six to eight days.

4. The *period of suppuration*. In this the vesicles pass into a pustular state and the umbilication disappears. The fever, which was low at the eruptive period, is again elevated and becomes more intense than before (fever of maturation). The duration of this period is from two to three days.

5. The *period of desiccation*. The pustules dry up gradually and become covered by yellowish crusts, changing to a blackish-brown, which are detached, leaving white, shining cicatrices or grayish-white spots. Its duration is from three to five days.

This is the normal progress of variola, but is not without deviations. The group of pustules may become united and form large, purulent patches (confluent variola); it is possible also that the disease may be complicated by a hemorrhagic diathesis (hemorrhagic variola); sometimes the skin is necrosed or gangrenous (diphtheric, fetid, or gangrenous variola).

Pathological anatomy. When we study with a microscope the successive variolous processes of the skin we notice that the cells of the mucous layer are swollen and present in certain points pale, non-nucleated bosselations. From the papillary bodies escapes a serum which dissolves the greater part of the cells; those which escape are stretched out into filaments by the exudate, the quantity of which augments without ceasing: thus they are found to constitute between the derma and the epiderma small cavities filled with liquid traversed by filaments and bands (vesicles). The papillary bodies and the subjacent layers are in a state of inflammatory tumefaction and of a round-cell infiltration.

The pustules are developed at the expense of the vesicles, by an abundant leucocytic diapedesis and the purulent disintegration of the walls. Healing is produced by desiccation of the pustules and by the absorption of the subcutaneous infiltrates. The epiderma is regenerated upon the cicatrices and borders.

Several theories have been advanced as to the mechanism which produces the umbilication of the variolic vesicles. According to some, this depression is due to the obstruction offered to the liquid by the hair follicles and the sudoriferous glands and tubes; according to others, it is the resistance of filaments and intra-vesicular bands; others, again, attribute it to an inflammatory infiltration more abundant in the periphery of the lesions than in their centre.

B. Variola of Domestic Animals.

I. CLAVELÉE: SCAB: VARIOLA OF SHEEP.

Etiology. Of all the variolas of animals, scab is the most important, because of the loss it causes to agriculture. It generally appears as an epizootic, and often extends over a very great extent

of country. Its infectious agent, fixed and *volatile*, possesses considerable resistance; in the sheepfolds it retains its virulence for five or six months; the convalescent subjects and the scabby animals may still communicate the infection for about six weeks. The virus of scab is rapidly and surely destroyed by dilute hydrochloric acid (Nocard), carbolic acid $1\frac{1}{4}$ per cent., solution of chloride of zinc or of quinine 5 per cent., solution of permanganate of potash 10 per cent. (Grunwald). The affection is ordinarily propagated by the introduction of diseased, convalescent, or scabby animals into the healthy flock; contamination may also be produced by intermediary agents (shepherds, dogs, clothing, wool, hides, manure, food, wagons, etc.). The receptivity for the contagion is general; the number of animals which escape is small. Lambs that are born of scabby mothers may possess immunity for a certain time or during their whole life, but sometimes they are born scabby. It may also happen that the fœtus dies in the uterine cavity, because of the disease.

This variola may be communicated, spontaneously or by inoculation, to the ox, pig, or man (Schmidt has described a case of contamination to man). Reciprocally, human variola and cowpox are transmissible to sheep. The direct introduction of the variolic lymph of man into the circulation of sheep produces a generalized varioloid eruption (Küchenmeister, Tappe).

STATISTICS In Germany, in 1886, this disease affected 4792 sheep; its principal centre is in eastern Prussia (Masmen), especially in the districts of Lyck Oletzko and Lotzen. We also find it in the district of Württemberg. In the same year it affected 1352 sheep in Austria, and 407 in the Pays-Bas. In 1887, in Roumania, it affected 64,000 animals.¹

Symptoms. After a period of incubation of from four to seven days on the average (minimum, 2-3), the disease is established. The animals are feverish, depressed, weak, they tremble, carry their

¹ In France scab-rot, which caused very serious losses in 1885-86, very notably diminished in 1887. For this latter year, of 37,430 sheep, composing the infected flocks, 17,352 were stricken with the disease and 2134 died (12 per cent.). The disease was observed in sixteen departments, but especially in the Lower Alps, the Higher Alps, Gard, Isère, and the mouth of the Rhone. Generally it is brought into the southeastern part of France by Algerian sheep. In this region, the southeast, in 1886, of 5650 inoculated sheep only 36 died—1 per cent. The mortality among the animals which have contracted the disease naturally is 14 per cent. (Tisserand: Rapport sur le service des epizooties en 1887).—N. D. T.

head low, cease to eat and to ruminate. The temperature is elevated, 41–42° C., and sometimes goes beyond these figures. The circulation and the respiration are very much accelerated; the conjunctiva is red, and there is a nasal and lachrymal discharge, but not abundant. Twenty-four to forty-eight hours later the regions covered with hair and those where the wool is not very thick (head, neighborhood of the eyes, the nose, the mouth, internal face of the anterior and posterior extremities, chest and belly, inferior face of the tail) present red points, then soon afterward papules of the same color. The exanthema is more rarely observed upon regions where the wool is abundant; sometimes we will notice several isolated pimples upon the buccal and pharyngeal mucous membrane. Ordinarily the cutaneous lesions produced by several successive developments do not present the same characteristics on the different regions.

On the fifth day the papules bleach in their centre, become vesicles, and are surrounded by a red zone; the skin of the neighborhood is markedly tumefied (head, surrounding the eyes), especially when the eruption is abundant. At the same time the fever falls. A few days after the centre of the vesicles becomes umbilicated and their dimensions gradually augment. Hemispherical, or more or less flat, they contain a lymphoid liquid, limpid and of a reddish-yellow color. At this period, six or seven days after the eruption, the vesicle reaches maturity.

If opened at this period, the virulent serum is seen to be cloudy, and becomes more and more so as the vesicle passes into the pustular stage. During this transformation we notice the symptoms of a general febrile infection, accompanied by localized catarrhal troubles. The fever goes up; the mucous membrane of the eyes, nose, pharynx, larynx, and bronchi are inflamed; the eyes and nasal cavities are the seat of mucopurulent secretions; the sick animals slobber, are seized with regurgitations, dysphagia, exacerbations of cough, the respirations are feeble; sometimes we notice a diarrhea; the head is greatly tumefied; the cutaneous perspiration is fetid. Finally the pustules dry up, the epidermis is retracted; the scabs, at first yellow, become a blackish-brown and fall off, leaving small depressions or cicatrices, which sometimes remain bare, but which afterward are thinly covered with wool.

When the subjects go through this normal course the total duration is about three weeks.

Course and Prognosis. It is quite a mistake to suppose that the evolution of sheeppox (scab-rot) is always thus regular. We sometimes observe a very benign form, with a sort of abortive course; the pustules exist in only very small number, the fever of infection is but slightly marked (discrete sheeppox). In others the eruption is perfect, the papules appear, become vesicular and pustular; then, in a few days, desiccation and desquamation (stony or warty sheeppox). Again, there are still others where the lesions do not pass into vesicles, and the exudation is very slight (flat sheeppox or scab-rot). [These divisions, eminently proper from the German standpoint, would not be of advantage to English-speaking veterinarians. A more simple and practical division is that which makes but two forms, *regular* and *irregular*, which very thoroughly covers all classes of cases.—W. L. Z.]

Among the serious forms of the disease we may specially mention confluent scab-rot. The groups of pustules unite and form abscesses or purulent masses of greater or less extent. We notice very large suppurating regions where the skin is in a state of intense inflammation and of considerable tumefaction; it may become necrotic and slough; then it exhales a fetid, gangrenous odor (fetid scab-rot). The fever is intense, pustules appear upon the mucous membrane of the mouth, pharynx, larynx, bronchi, and also upon the cornea. The lymphatic ganglions of these organs are greatly tumefied, and sometimes they contain purulent centres; the inflammation of the mucous membranes may assume a croupous character. Pneumonia is frequent. When the disease has a fatal termination we observe the symptoms of septicemia and pyemia, and metastases in the articulations, serous membranes, brain, etc. The animals die from pneumonia, from asphyxia produced by the laryngitis, and from infectious complications. In this form, when recovery takes place, the convalescence is prolonged. The subjects are exhausted, cachectic; sometimes the skin is entirely denuded and marked with deep cicatrices; frequently both eyes are lost; often, also, there remains a chronic lameness.

In hemorrhagic variola the eruption is accompanied by cutaneous and mucous hemorrhages, and the pustules undergo gangrenous destruction (diphtheric, gangrenous scab-rot). We also notice hematuria.

The course of the epizootic in the flock is sometimes very slow, at other times rapid. Sometimes it continues for a long period. It

may have a benign form in certain of the sick animals, malignant in others; the number of animals which escape is very small, 2 to 3 per cent. When there is a regular evolution of the disease the mortality is from 10 to 20 per cent.; in the serious forms it is 50 per cent., sometimes greater. In the confluent or hemorrhagic forms, or when the affected animals are old and feeble or very young (sucking lambs), the prognosis is always unfavorable; it is aggravated by various circumstances: rainy weather, cold, humid or too much heat, irrational food, or defective sheepfolds. Sheep recently imported into a country whence the disease exists are more seriously affected than the indigenous races which are acclimated, a certain number of which have already been affected.

Abstracts of the mortality caused by scab-rot show that it still exerts a prejudicial influence upon agriculture. The animals lose their wool; they are greatly reduced in flesh; many of the sheep abort; convalescence is very long, and often they continue in a persistent morbid or diseased condition consecutive to this disease.

Treatment. As in all the acute exanthemas, the treatment is purely expectant and dietetic. When complications supervene we may apply a symptomatic treatment; but this is difficult to carry out in practice because of the great number of sick animals. The prophylaxis comprises inoculation and sanitary police measures.

Inoculation (ovination). Preventive inoculation for scab-rot was practised on a very large scale during the first half of this century up to 1860. Special institutions were established by which to facilitate and to extend this operation, which we carried out each year upon a number of flocks, even when there was nothing to make us suppose that an epizootic was approaching. We have since then recognized that this procedure was a dangerous one and have abandoned it. The inoculated animals easily contaminate the healthy ones. By inoculation we create permanent centres of infection in which the disease becomes stationary and from which it radiates into the surrounding regions. In some countries (Prussia, Austria) the extension of scab-rot was in proportion to the preventive inoculation.

On the other hand, *inoculation of necessity* is recommendable. It is contained in the law of sanitary police. This operation consists in inoculating the animals of infected flocks. By the inoculation of necessity the duration of the epizootic is shortened and the disease is much less serious than spontaneous scab-rot. The mortality

of inoculated scab-rot is often *nil*; ordinarily it is about 2 per cent. We have seen losses of 10 per cent., but only when the external conditions are very unfavorable.

Inoculation, *preventive* or *precautionary*, may be indicated in a certain region when the scab-rot is progressing with marked intensity in the neighborhood; contamination of the flocks is inevitable.

TECHNIQUE OF INOCULATION. We draw the "lymph" from the subjects which have a benign inoculated scab-rot and which present well-developed pustules. The lymph should be perfectly limpid and transparent; it is obtained ten or twelve days after the inoculation, or six to eight after the eruption. The animals are then isolated; we must remove them from the sheep that have furnished the virus, as spontaneous infection may take place notwithstanding the inoculation.

This operation is practised upon the internal face of the ear about 4 centimetres from the point, or the inferior face of the tail 10 or 12 centimetres from the anus; in the latter case the animal has to be laid down. We may use a lancet or a straight needle, making a puncture or excavation in the form of a quill. When it is necessary to inoculate an entire flock it is advisable to operate upon six or twelve individuals at a time, unless the circumstances of the case oblige a more rapid procedure.

On the inoculated sheep we observe a variolic exanthema localized at the seat of the operation; the general symptoms are but slightly marked. The pustules reach maturity about the twelfth day. In exceptional cases they do not appear at the point of inoculation, but in the adjacent regions. It is still more rare that the local exanthema produced by the inoculation is followed by a generalized eruption. The inoculated animals should be sheltered from inclement weather and given good food. Ten or twelve days after the operation we examine carefully the entire flock and make a new inoculation of those which did not respond to the first.

Of late years attenuation of the virus has been the object of experimental research. Pench attempted to obtain this by dilution of the lymph in ordinary water: 1 part to 50-150; Nocard and Mollereau employed oxygenated water; Semmer-Raupach employed 55° C. of heat; Toussaint and Plaut cultivated the virus in different liquids. According to Pourquier, inoculation of the lymph taken from a pustule after the tenth or twelfth day, employing the ordinary method, confers immunity without producing the symp-

toms of scab-rot. This author recommends inoculating at the base of the tail.

II. VACCINE, COWPOX.

Etiology. The opinion of Jenner that cowpox was produced from variola of the horse does not find any supporters at the present day. On the other hand, Turner, Leroi, Ceely, Sunderland, Thiele, have indicated the relations which exist between the variola of the cow and that of man. Roloff and Bollinger have established by indisputable facts the etiological connections which exist between these two diseases. According to Bollinger, the actual conditions are that cowpox is not proven to be the true human variola, as has been asserted by some, but rather a vaccine transmissible to man. This opinion is supported by the frequent coincidence in the spring of the vaccination of children and epizootics of cowpox observed upon the cow, the numerous ways of contamination of this latter by human vaccine, and the facility of experimental transmission of vaccine to the ox.

The virus of cowpox is fixed; the infection always takes place by a wound of the skin, as well as that which is produced at the time of milking. The result of experiments by Chauveau, Warlomont, and Hugues, show that subcutaneous and intravenous injections do not determine local or general eruption, although they confer immunity. Transfusion to a healthy calf of blood from an animal of the same species affected with cowpox confers immunity from the first without producing appreciable morbid phenomena (Reynaud). Cowpox may be communicated to the sheep, the goat, horse, and to man. Reciprocally, the variola and vaccine of man are transmissible to the ox. The inoculation of variola or vaccine of man to bovines generally gives immunity to these animals without producing manifest disturbances. Sometimes they result in a specific eruption (Sunderland, Dinter, Woodwille).

In Würtemberg, from 1825 to 1868, there were 241 cases of cowpox; and from 1873 to 1878, 100 cases. In Denmark, during the year 1874, there were observed 374 cases, and in 1877-78, 1037 cases. As a general rule, this is a rare affection.

Symptoms. Cowpox is observed more especially upon young fresh milking cows; ordinarily the exanthema is localized upon the teats, the udder, and the neighboring regions; the fever is *nil* or very slightly marked. The eruption takes place by successive

crops. The diseased animals manifest only light general troubles, but the secretion of milk diminishes. The milk itself is more watery, its specific gravity is below normal. The affected teats are generally tumefied and sensitive; we also notice upon their base a number of isolated pimples (twenty to thirty), rose-colored, and from the size of a lentil to that of a pea, which are transformed into umbilicated vesicles having a color varying with the thickness of the skin. If this is white and very fine, they are of a bluish-white color and a mother-of-pearl reflection; when this is of a clear tint and thin, then they are a brilliant red; if it is of a dark color, they have a leaden reflection; lastly, when it is thick and colorless, they are in turn grayish in color. When they are developed upon the teats they are elliptical in form; upon the udder they are round. When the skin is non-pigmented, they are surrounded by a reddish areola; they are always circumscribed by a tumefied zone. They reach maturity about the tenth day, when they are almost the size of a kidney bean. At this stage of their development they become conical and suppurating (pustules), then they dry up and become covered with a dark-brown crust, which falls off in about four days, leaving a smooth and brilliant cicatrix. Often these vesicles are torn during the manipulations of milking. The total duration of the exanthema is about twenty days. In a few rare cases where they are generalized we find vesicles on the head and on the internal face of the thighs, etc. In the bull we have observed them upon the scrotum. The prognosis is favorable, and all treatment is inefficient.

In the same individual cowpox gives several successive eruptions of pustules presenting divers degrees of development. In the cow-houses the disease is propagated slowly from the first animal to all the others; it is rare that any cow escapes; the bull, the ox, and the young animals most often escape; in them the contamination constantly takes place through the litter. Generally the epizootic persists for several weeks in the same stable.

Differential diagnosis. Cowpox must be distinguished from aphthous fever, bovine plague, eczema of grains and refuse, and mercurial poisoning (mercurial ointment used in cases of mammitis). It must also be differentiated from:

1. *False cowpox* or *varicella*, still known as *aqueous* or *gaseous variola*, an affection characterized by vesicles from the size of a pea to that of a cherry, and which gives birth to thin crusts.

2. *Warty variola*, indicated by multiple papilloma of the teats, lesions which persist for a month, disappear slowly, and are not at all contagious.

GENERAL CONSIDERATIONS OF ANIMAL VACCINE IN MAN. After Jenner's discovery in 1798 we used exclusively, for the inoculation of man, the *original* or *true vaccine* lymph coming from the cow. Because of the difficulty of procuring the vaccine, on account of the rarity of cowpox, we made use later on of *humanized* vaccine, or a vaccination from man to man. The possibility of inoculating at the same time as the vaccination other diseases of man (syphilis, tuberculosis, etc.) caused physicians to return to the animal vaccine. In order to obtain the lymph, vaccine institutes were established at Milan, Paris (1864), Brussels (1863), and more recently in all the large cities. We cultivate the vaccine upon calves from five to twelve weeks old (more rarely we take a calf of six months, the heifer, or the cow). The technique of the operation is simple. We cast the animal upon a table, on the left side, and place the superficial posterior member in a vertical position. We shave the skin of the abdominal region from the symphysis pubis to the umbilicus as far as the flank, then we wash thoroughly. We then practise the inoculation by making small parallel incisions or from 50 to 200 pricks with a lancet. Vaccine from a calf previously inoculated is placed upon the wounds thus produced.

The pustules which are developed reach their maturity in about four or five days; then we gather the lymph destined for the vaccination of man and for other cultures upon the calf. This is done by the application of pressure forceps upon the base of the pustules. A single calf will furnish enough lymph to vaccinate from 1000 to 3000 subjects. In order to preserve the lymph we may choose from different methods. Capillary tubes are not to be recommended; the lymph loses its virulence in about one week. It may be preserved better in a dry state: we commence by collecting the serum and the scab, we detach the whole mass, which is placed between two plates of glass and sealed with paraffin; in these conditions the vaccinal material remains active for months, even for years. We may also mix the lymph with glycerin and add to the mixture an antiseptic. It is also prepared by aspirating into capillary tubes which are afterward closed. We employ in preference the solutions: thymol, 0.1; alcohol, 0.5; glycerin,

100; or, salicylic acid, 0.25; distilled water and glycerin, āā, 50 (Ropke).

Vaccinal exanthema is developed very much better by the inoculation of cowpox than by the vaccination of man to man. We should give preference to the first operation. We can procure animal lymph in any quantity, and we are not likely to transmit the diseases which are sometimes communicated by human vaccine. With the lymph recultivated upon the calf we are quite certain, so to speak, of not inoculating tuberculosis; this disease is, in fact, extremely rare in very young individuals of the bovine species, and never have we been able to demonstrate this transmission by animal vaccine. Besides, we can surely avoid this accident by submitting the calf to a complete examination, and not employing for the cultures of vaccine any but perfectly healthy animals.¹

III. VARIOLA OF THE HORSE: HORSEPOX.

Etiology. In former times variola of the horse was known by the name of "preventive leg grease," "true leg grease," "exanthematosus." It is very rare at the present day (?). According to H. Bonley, it may be seen quite frequently in the environs of Paris. If horsepox and cowpox are derived from human variola, it is very probable that the equine lymph would be identical with the bovine lymph or vaccine. In the inoculations of the cow we produce lesions similar to those of vaccine, and we confer on this animal immunity against the variola of man. Experimental inoculation (Hertwig and Pingaud) and the accidental transmission (observed in horseshoers and hostlers) produce in man a benign vacciform eruption. Bollinger admits that variola of the horse is generally localized on the pastern, because this region is very often the seat of wounds and scratches. We think that the inferior part of the extremities is the preferred seat of the variola

¹ The question of determining the transmissibility of tuberculosis by the insertion of vaccine coming from tuberculous subjects is still the object of experimental research. In 1881 Toussaint announced to the Academy of Sciences that he had rendered tuberculous two rabbits and a pig by inoculating them with vaccinal serum from a tuberculous cow. Lothar, Meyer, and Guttman have searched in vain for the bacilli of Koch in the contents of vaccine pustules developed upon tuberculous animals, and Straus, Chauveau, and Jossraud, who have repeated the experiments of Toussaint, have obtained only negative results. No one has ever reported an authentic instance showing the transmission of tuberculosis by animal vaccine. To remove all danger we advise an autopsy upon all animals from which the vaccine is taken, in order to show that they are not tuberculous.—N. D. T.

exanthema, because it is so frequently in contact with the hands and arms of man (shoeing, grooming, etc.), parts of the body upon which vaccination is usually performed. From the point of view of the chances of contamination, the pastern of the horse offers about the same condition as the udder of the cow.

Very rarely is variola of the horse generalized. Chauveau has produced this form by inoculating the virus into the veins under the skin, and by causing it to enter the organism by inhalation or by ingestion. Warlemont and Pfeiffer have obtained similar results. The horse may contract sheeppox, cowpox, and the variola of man.

Symptoms. Horsepox is indicated by fever, by depression, and by loss of appetite. Upon the surface of the posterior face of the pasterns an erythematous exanthema is developed, indicated by tumefaction and redness of the skin, which extends more or less high; the diseased animals limp, stamp, lift high these members, and carry them in abduction. On the affected regions we observe vesicles and pustules which are rapidly ruptured, the skin becomes bare and greatly tumefied, red and painful, coated with a viscid exudate, which soon dries, forming a large crust. In a few weeks recovery is complete. In certain rare cases the exanthema appears on other regions where the hair-covering is not very abundant: as on the nose, the lips, nasal and buccal mucous membranes, and the conjunctiva.

Differential diagnosis. The existence of the variola of the horse is not to be doubted, although very often it has been confounded with other eruptive affections. In a number of cases observers have taken for horsepox pustular contagious stomatitis, with which it offers a great analogy, excepting the localization. The confusion is so much easier, as exanthema of this form of stomatitis is transmissible to the ox accidentally and by inoculation. From an etiological standpoint, however, these two diseases are different.

Moreover, *coital exanthema* and the *ordinary traumatic scratches*, the lesions of which have nothing regular in their course, and which are not transmissible by inoculation, may also simulate horsepox.

IV. VARIOLA OF THE PIG.

Etiology. Variola of the pig seems to be the result of an infection having a human origin (beds of straw occupied by variola

patients) or by sheep (keeping pigs in sheepfolds that are infected with scab-rot). It is ordinarily observed upon young animals. Gerlach succeeded in inoculating the variola of the pig to the goat, and reciprocally. Man may also contract it.

Symptoms. The eruption is habitually generalized. The animals are feverish, very weak, cease to eat; the mucous membranes are red. Upon the head, neck, back, chest, and the internal face of the anterior and posterior members we observe red points, with the successive development of papules, vesicles, and pustules, which desiccate. These are soon covered with rounded, concave, black crusts. In some rare cases the variola exanthema is noticed upon the buccal mucous membrane. The intensity of the disease is very variable. We may often confound it with urticaria and pustular eczema.

V. VARIOLA OF THE GOAT.

Variola of the goat is rare; special *bibliography* only mentions a few observations. According to Brémond, this affection constitutes a form differing pathologically from scab-rot; it is not inoculable to sheep, and scab-rot cannot be communicated to the goat, either by inoculation or by cohabitation. Sometimes caprine variola shows the character of a generalized scab-rot; at other times it assumes those of an exanthema of the mammae, having all the symptoms of cowpox. In the goat we also have successive crops of eruption (Hertwig).

VI. VARIOLA OF THE DOG.

Human variola may be communicated to the dog by inoculation (Dupius and others). In a few cases it has been transmitted accidentally (Weiskopf).

Certain observations reported of "variola of the dog" appear to be related to other affections; it is probable that they have taken for variola, pustular exanthema of canine distemper, the "eruption of acaries" (follicular mange), and perhaps aphthous fever. In any case the predisposition of the dog to variola is very slight.

The clinical cases designated as *variola of chickens* are related to epithelioma of gregarina. The experiments of inoculation of variola to chickens have given only negative results (Gunther, Hurtrel, d'Arboval, Rayer, Plaut, and others).

BOVINE PEST: CATTLE PLAGUE.

HISTORY. Although ancient medical literature is silent as to bovine pest, this disease must have existed in the steppes of Oriental Europe and of Central Asia since the most remote periods. The first documents collected upon it are contemporaneous with the appearance of people in those regions, as universally shown in history. By the human migrations of the fourth century of our era—the Huns coming from Central Asia, the Alains from the steppes bordering upon the Volga, the Ostrogoths from Southern Russia, the Visigoths from Northern Hungary—it was imported into the States of Western Europe. It soon spread throughout the Continent, owing to the incessant wars of the period. In the ninth century, under the government of Charlemagne, it ravaged Germany. At the commencement of the thirteenth century the invasion of the Mongolians again spread it over Eastern and Central Europe, where its devastation caused considerable losses. In the course of the eighteenth century it caused immense losses (Northern wars, Successions wars, and Seven Years' war). The first great epizootic prevailed from 1709 to 1717. Beginning in Tartary, it was propagated along the rivers Don and Volga, reaching Moscow, then invading in succession Poland, Hungary, Prussia, Austria, Southern Germany, Switzerland, Italy, France, Holland, and England. In the years 1711 to 1714 it caused the death of 1,500,000 cattle. At this period the first sanitary measures were instituted against it. Ramazzini (1711) gave a very exact description of this disease, which he compared with variola. Buniva has recorded the march of the epizootic of 1726 to 1734.

From this latter date up to the commencement of the nineteenth century this pest remained stationary in almost all the countries of Europe. Spain and Sweden have been preserved from this disease, because they did not import any foreign cattle. We estimate the number of animals that succumbed to it from 1740 to 1750 at three millions. In Denmark alone there perished more than two millions from 1745 to 1752. Toward the end of the last century bovine pest caused the death of thirty millions of cattle in Germany, and in the whole of Europe two hundred millions, representing a value of more than seven billion dollars. It was

these enormous losses which caused the government to establish veterinary schools. Monographs relating to the pest multiplied rapidly (Bomgelat, Bonhave, Wollstein, Layard, etc.). Lutz, in 1783, collected in the neighborhood of one thousand articles concerning this disease. Then we already practised a preventive inoculation.

First practised by Dobson, of England, in 1744, this operation was introduced into France in 1745 by Courtivion; little by little it expanded into all countries. It was experimented with by Camper and Grashnis in Holland, Oeder and Viborg in Denmark, Bülow and Oerzen in Mecklenbourg, Kersting in Hanover, Adami at Steiermark, Nebel in Hesse, Reich in Franconia, Sick in Prussia, Namsler in Silesia, Lorinser in Galicia, Pessina in Italy, Barrasch in Hungary, Walz in Würtemberg, Jessen and Raupach, then Sergejew and Kobischew in Russia.

At more recent dates bovine pest has again caused very great losses. In 1792, in Italy, where it was introduced by the provisioning of the Austrian army, 3,000,000 to 4,000,000 of cattle perished. From 1795 to 1801 it was especially severe in Southern Germany. The wars at the commencement of this century (1805–1809–1813–1816) were particularly favorable to its propagation. A new European invasion, which started in Moldavia and Valachia, existed in 1827–28; in 1830–31 (insurrection of Poland); another came from Russia from the provinces close to the Baltic Sea. In 1841 oxen which came from Roumania and Austria introduced it into Egypt, where it killed 500,000 animals; from 1844 to 1845 Russia lost 1,000,000 of cattle; then it appeared nearly every year in Austria and Prussia. According to Röhl, from 1847 to 1864, it killed nearly 500,000 animals in Austria. It also appeared during the wars of 1866 and 1870–71. In 1870 it killed 100,000 oxen in France (30,000 in Alsace-Lorraine), and 10,000 in Germany. The last epizootic observed in our country (1878–79) caused losses estimated at \$500,000. Prussia lost 2500 animals, and Russia 350,000 at the same period. In general, bovine plague seems to be extinct in Germany, and this is due to the active watchfulness which is exercised upon its frontiers and to the strict application of the measures which are prescribed by the law of sanitary police.

Etiology. Among experimenters who have made bacteriological researches upon the infectious agent of bovine pest we may men-

tion Sanderson, Bristowe, Murchison, Beale, Semmer, Naczynski, Hallier, Klebs, Roschnow, Woronzow, Medwedski, Laweljeff, Metchnikoff, and Gamaleia.

Semmer considers the infectious agent to be a streptococcus, which he inoculated and cultivated in peptonized gelatin, where it formed a sort of pale grayish-green growth, liquefying the culture medium.

Laweljeff has cultivated spore-forming bacilli which were transformed into isolated micrococci or arranged in chains. Upon agar the cultures at first have a pale grayish color, which afterward becomes a citron-yellow, and finally red. Inoculations of these cultures produce bovine pest. The bacilli take the methyl-violet stain; they are round on their extremities, and are endowed with special movements; in the blood they are sometimes straight, sometimes curved; when we examine them directly, without staining, they are difficult to distinguish. Laweljeff thinks that the investigators who preceded him saw the same microbe, but in another stage of evolution.

Metchnikoff has found a short bacillus with rounded extremities, which may also present the appearance of a micrococcus or a leptothrix. We find them in preference in the ulcers of the stomach and in the blood. They grow well in gelatin without liquefying it. By the inoculation of cultures of this microbe, Gamaleia has produced in the calf and guinea-pig (but not in the rabbit) an affection analogous to bovine pest.

Pathology. Semmer asserts with a certain degree of reason that the infection is produced through the respiratory organs; from there the contagion penetrates into the blood, and the disease becomes generalized. The lesions of the digestive apparatus are of secondary development.

The contagion is fixed and *volatile*. It exists in the secretions and excretions, in the excrements, urine, saliva, mucous secretions of the nose, mouth, and eyes, in the sweat, expired air, blood, and in all the tissues. Contamination may take place directly by the diseased animals, or indirectly by the dung, the bedding, the hay, the earth, hides, wool, meat, clothing, wagons, vessels; by men (butchers, dealers in animals, smugglers); by dogs, sheep, and chickens, etc. The virus is not very subtle; contagion takes place only through relatively short distances. In summer, when the weather is dry, this is reduced to its minimum (about 25 metres),

and the progress of the epizootic may be stopped by a ditch separating the diseased from the healthy animals. The infectious agent seems to be rapidly destroyed by air, especially by dry air. It resists for a long time when it is contained in liquids or in solid matters. In the nasal mucus, placed so as to be protected from the air, it remains active from six weeks to nine months. It may retain its vitality for four months in the stables, for five months in hay. We may produce bovine pest with cadaveric remains which have been buried for nine months; dung frozen through the winter is found to be infectious in the spring after thawing. The contagion is destroyed by temperatures of $+60^{\circ}$ and -15° C., by putrefaction, by various chemical agents: chlorine, sulphuric acid, carbolic acid, etc., and by desiccation. It is rapidly destroyed by most disinfectants. It possesses its maximum of virulence at the onset of epizootics.

AFFECTED ANIMALS. Bovine pest has been designated by the terms *contagious typhus*, *obstruction of the omasum*, *disease of the omasum*, *cattle plague*, *contagious disease of cattle*, *epizootic disease of horned cattle*, etc. This is an infectious disease especially severe in the ox, but transmissible to other ruminants (stag, yak, antelope, gazelle, chamois, buffalo), and in the wild boar (Penning). A first attack confers immunity. We do not yet know exactly the regions which constitute its primary centres, or the cradle of typhus. Sergejew and Semmer pretended that it originated upon the steppes of European Russia (Southern Russia, the region of the Black Sea, and the territory of the Don), where the surface of the soil is formed of a thick layer of mould, and where vegetation is very luxuriant. According to others it had its origin in Asia, either in Siberia, on the steppes of the Kurdistan, or Tartary, China, Persia, India, etc.—that is to say, outside of European Russia, and very probably in Central Asia. However this may be, it is demonstrated that the steppes of Russia or of Asia are the centres of transmission. The epizootic which appeared in England in 1865 was imported by oxen coming from Russia. That which appeared in Switzerland in 1866 had, as its first cause, transportation of Austrian cattle, and, a second, of animals bought in Russia. The countries adjacent to this latter country—Germany and Austria—are constantly threatened by the plague. Formerly the extension of this disease was feared only during the period of wars (provisions for the army); it is now the result of commerce, of transports, of fairs, etc., and sometimes by

the intermediation of infected products: hides, meats, wool, butter, grease, etc.

Symptoms. The symptoms of bovine pest are those of an acute, serious infection with localization upon the digestive apparatus. The period of incubation appears to be from six to nine days on the average. Boloff and other authors have noticed an elevation of temperature from thirty-six to forty-eight hours after infection. Raupach and Bavitsch have noticed pronounced anatomical alterations after thirty-three hours, also eleven hours after inoculation; after seven hours Semmer found in the blood and nasal mucus the micro-organisms which he has described. The first symptoms are those of fever, which may easily pass unperceived; he also admits that the incubation period is short; it does not exceed from three to six days.

The disease is indicated by certain prodromes, especially by an intense hyperthermia; the rectal temperature is elevated, 41° to 42° C., and more. The fever, continuous, presents short remissions. As in other infectious maladies, there is short thermic depression at the moment when the local symptoms appear. The pulse is very weak, and from 60 to 120 per minute. Besides these febrile phenomena we observe a general weakness and a diminution of the mammary secretion (according to Gerlach and Bruckmüller, agalactia is often the first trouble apparent); the hair is staring and the muzzle dry, the appetite is diminished, the rumination suspended, and slight tremblings are noticed in different regions.

These prodromes are followed by manifestations which characterize the *symptomatic period*. The sick animals are seized with a chill, the temperature of the surface of the body varies, the respiration is accelerated, the visible mucous membranes (conjunctiva, pituitary, buccal, anal, vaginal) are red and spotted as in scarlatina. The loss of appetite is absolute, rumination is arrested, the thirst intense, defecation retarded; the excrements are dry and coated with mucus; often there are slight colics. There is a serous discharge, then sero-mucus, from the eyes, nose, and vagina; the salivation is abundant. Little by little the excrements are softened, and we observe signs of abdominal pains and a hemorrhagic diarrhea, colliquative and fetid; there is a tenesmus and sometimes a rectal prolapsus. They loose flesh very rapidly, the walk is staggering, the lumbar region is sensitive to pressure or palpation. The majority of the animals remain recumbent, some are restless, excited,

and present symptoms of a rabiform character (cerebral congestion, nervous form of bovine pest); in others we notice marked dyspnoea and the ordinary signs of pneumonia, especially the cough, the râles, and the dulness (pneumonic form).

The characteristic alterations soon appear upon the mucous membrane of the lips, tongue, cheeks, gums, nose, and vagina; we may notice red spots disposed in patches or bands, which are soon converted into sloughs or eschars of a whitish-gray, and but slightly adherent; the epidermic layer is thickened and presents grayish-yellow points. More rarely the eschars are preceded by pimples, which rapidly undergo caseous degeneration; when detached they leave depressions, which are dark-red and bleeding (erosive ulcers). In benign cases the eschars and the ulcerations may be absent. Sometimes we observe on the skin a process resembling exanthema (ancient exanthematous form of bovine pest), upon the abdomen and the internal face of the thighs, the perineum, and the mammæ; this membrane is covered with small pimples and pustules and their scabs. In certain cases when these lesions are localized upon the mammæ they may be mistaken for cowpox. Pregnant females abort in great numbers.

When the disease is to have a fatal termination loss of flesh and exhaustion become more and more accentuated; the sick animals remain extended upon the earth, trembling, grinding their teeth; the natural openings allow the escape of fetid liquids; the anus and the vulva are open; the temperature is below normal. Death supervenes in coma or convulsions. During the death struggle there is an escape of bloody, frothy mucus from the mouth and nose.

Course and Prognosis. Ordinarily death occurs in from four to seven days; in more serious cases it may not be more than five days. The symptoms may attenuate gradually, and recovery take place. Convalescence is quite long. In large herds of cattle, or where there are many cow stables, the epizootic has a slow progress, and advances only by *fits* and *starts*. At first it affects only one group of animals, placed in one position or another, and its generalization always requires several weeks.

The course of bovine pest varies with the epizootics and the breed of the affected cattle. At the time of the invasion of the epizootic the disease is more serious than toward the end. The races of the steppes (Southern Russia, Hungary, Roumania, Servia, and Mol-

davia) possess a (partial) hereditary immunity. The plague may, therefore, present itself in a relatively benign form and with symptoms of moderate intensity; often followed by recovery in about eight days. In the animals of Western Europe the mortality is 90 to 95 per cent.; in the gray races of the steppes it is only 30 to 50 per cent.

In the sheep and goat the symptoms are the same as in the ox, but the affection is less grave and the contagion less subtle. In Austria (1859-63), in 4000 sheep affected by the pest the mortality was 66 per cent. In Hungary, 1861-63, it was very nearly the same; in Russia, out of 30,000 sheep affected in 1878, it was 60 per cent. Contrary to what exists in the ox, pulmonary localizations are frequent.

Differential diagnosis. When bovine pest appears in a region in which it claims only a few victims the diagnosis often presents great difficulties. In this respect the most important symptoms are the intense hyperthermia, the red spots, and the yellowish-gray exudate of the mucous membranes, the ulcerous erosions of the vagina, the grave intestinal troubles, and the marked emaciation. The possibility, the probability, or the certainty of a contamination is of the highest importance.

Among the diseases presenting symptoms that may be confounded with bovine pest we must particularly mention the following:

1. *Malignant catarrhal fever.* This disease has a contagious character very little accentuated, a slow course, and a localization preferably upon the head and respiratory organs; generally it is stationary in certain regions; the serious ocular trouble may guide the practitioner in the majority of cases.

2. *Apthous fever.* This sometimes presents a certain analogy with bovine pest in the ulcerations of the buccal cavity, the gastric disturbance, and the mammary eruption. But the exanthema of the mouth, sufficiently characteristic, coexists with lesions of the same nature localized at the coronet and in the interdigital space; moreover, the epizootic is benign and is propagated very rapidly.

3. *Dysentery.* In this disease the diarrhea is often of greater severity than in bovine pest; the intestine alone is affected; the other mucous membranes, mouth, eyes, nose, vagina, are intact. The alterations found at the autopsy are different.

4. *Mycotic enteritis.* That which is produced by the *Tilletia caries*, for example, may have a great resemblance with cattle plague. Very

serious, it often appears as an enzootic. Here, again, the intestinal disease predominates, and we may observe nervous symptoms.

5. *Anthrax (charbon)*. Its gastric form may be confounded with bovine pest, but its course is more precipitate and more alarming than this latter; it is only exceptionally transmitted by direct contagion; at the autopsy it is easy to establish a diagnosis by finding the bacteridium.

6. *Pleuro-pneumonia* is marked by symptoms elicited by an exploration of the chest; besides, certain symptoms of typhus are wanting (a phlegmasiac pleuro-pneumonia may be developed in the course of the latter).

7. *Hydrophobia* and *cowpox*, which are, as a rule, easily recognized.

Pathological anatomy. The anatomo-pathological alterations of bovine pest are principally located upon the mucous membranes of the abomasum, the intestinal grêle, the mouth, the rectum, and the vagina. The general lesions similar to those observed in other contagious diseases offer but little importance.

The cadavers are emaciated and the posterior members are soiled by excrements; the margins of the mouth, the nostrils, the eyes, the anus, and the vulva are covered with a yellowish muco-purulent matter. Sometimes we notice pimples and pustules upon the skin, especially on the mammæ. The buccal and pharyngeal mucous membranes are strewn with red and tumefied spots, which are covered either by a grayish-yellow caseous exudate or by a layer composed of cellular detritus, of nuclei of micrococci, and produced by a superficial diphtheric inflammation. By raising these false membranes we discover ulcerated red wounds (erosions). These alterations, are especially marked on the inner fascia of the lips, the cheeks, the lower portion of the tongue, and on the gums (inferior jaw).

The mucous membrane of the first three gastric compartments is spotted red; the epithelium is tumefied and dropsical and is easily removed; the contents of the paunch and reticulum are dry; that of the omasum is sometimes normal, at other times completely dried (obstruction of the omasum), but this lesion is common to a number of diseases. Sometimes the abomasum is empty; more rarely it contains a small quantity of yellowish or bloody muco-purulent viscous liquid; in the neighborhood of the pylorus the mucous membrane is hemorrhagic and ecchymosed, varying in color from a

violet to a cherry-red, or approaching a brown-red, showing a slaty reflection. It is covered with small islands of a yellowish-brown caseous exudate, which become detached leaving in their place red erosions. The epithelium is desquamated. The gastric glands (pepsin and mucous glands) are tumefied by an abundant cellular infiltration.

Similar alterations exist in the intestinal grêle. Here also the mucous membrane is greatly inflamed, very red, and covered by a caseous exudate in patches resembling superficial eschars. In serious cases the intestine contains a cylindroid exudate completely covering its surface. The solitary follicles and Peyer's patches are greatly infiltrated. These organs are prominent, surrounded by a red areola, and undergo a purulent degeneration (by compressing them drops of pus escape); sometimes they are covered by a caseous or pruriform layer. On detaching these exudates there is exposed an ulcerating surface. The Peyer's patches often have an areolated appearance; the glands of Lieberkühn are tumefied; the intestinal villi are the seat of cellular infiltration. According to Klebs, the entire mucous membrane is overrun with micrococci, which are especially accumulated around the bloodvessels.

In the large intestine the inflammatory alterations are much less pronounced, nevertheless the cæcum is very seriously affected; as a rule, the mucous membrane is tumefied, of a slaty color, with red spots, and covered with mucus. In some cases these lesions, slightly marked, are also observed upon the intestinal grêle. It may be that this latter is entirely necrosed.

Often the mesenteric ganglions, which are voluminous, have an encephaloid consistency; sometimes we do not observe any other alterations. The liver, which is pliable, has a clayey color; the biliary vesicle is distended as a consequence of obstruction of the ductus communis choledochus (from whence the names of "grosse bile," "surbile," formerly given to bovine pest); the mucous membrane of this sac is congested, red, and covered with yellowish-gray exudative patches. The kidneys are marked by parenchymatous alterations; they have a brownish-yellow coloration and a friable consistency. The mucous membrane of the bladder is red and coated with mucus; that of the uterus presents similar alterations, which are usually more pronounced; it is the same with those of the vagina and rectum.

The nasal mucous membrane is dark red and covered with yellowish-gray friable crusts. We notice similar alterations in the

larynx and trachea, where the exudative masses often have a creamy or purulent consistency. Sometimes the lungs are congested and strewn with hemorrhagic spots; at other times they are œdematous, hepatized, or emphysematous. Pneumothorax and subcutaneous emphysema are rare. The heart is flabby, soft, and infiltrated with ecchymoses. The endocardium is of a blue or red shade; under this serous membrane, and under the epicardium, we find hemorrhagic centres; the pericardium contains a yellowish transudate. The blood has a dark coloration, it is imperfectly coagulated, the number of white globules is augmented, and the red globules present various alterations of form (poikilocytosis). The nervous centres and their envelopes are hyperemic; we may find a reddish transudate in the cerebral ventricles and under the arachnoid.

These lesions are far from being always equally marked; they vary with the character and intensity of the process, the state of general nutrition, and the race and age of the patients. In animals of the gray species of the steppes (the same as in the sheep) they are relatively little pronounced.

Treatment and inoculation of bovine pest. The treatment of bovine pest is very inefficient and offers but little interest. The law upon this contagious disease is positive, from which there is no appeal; it prescribes immediate slaughter. In certain countries, especially in the regions of the steppes, inoculation is *necessary* as an important prophylactic measure. Preventive inoculation should be absolutely rejected on account of the dangers of propagating the epizootic. In all the countries of Europe, with the exception of Russia, inoculation is not advisable; it results in a high rate of mortality. Slaughter is much to be preferred. In the race of the steppes the mortality consecutive to inoculation is 10 per cent.; in the others it reaches 36 per cent. It is in the declining period of epizootics that the results are most favorable.

The method of inoculation is simple. In order to gather the virus we tampon the nasal cavity by means of a clean sponge; we wait until this is completely saturated with mucus, and we collect it in tubes, which are afterward hermetically sealed(?). The operation consists in injecting, by means of a syringe, one drop under the skin of the neck. But even in Russia, where there formerly existed four institutes for the inoculation of bovine pest (Charkow and Karlowka, under the government of Poltawa;

Boudarewka, under that of Cherson; and Salmysch, under that of Orenbourg), this practice is entirely abandoned at the present day. Recent discoveries made in regard to the prophylaxis of infectious diseases leads to the belief that we may succeed in attenuating the virus of bovine pest, and by it create immunity without suffering the great losses produced by inoculation as practised up to the present time.

CONTAGIOUS PUSTULAR STOMATITIS OF THE HORSE.

Nature. Contagious pustular stomatitis of the horse is an acute infectious exanthema with benign course, marked by pustules which are especially developed upon the buccal mucous membrane.

HISTORY. This disease has been known for a long time. In 1840 Dard described a vesicular inflammation of the nasal mucous membrane; in 1832 it was observed upon fifteen horses of the same troop, and was first taken for acute glanders. Its course is benign, and lasts about twenty days. H. Bouley, Patté, Reynal, Jacob have mentioned similar facts. Later, in 1856, Hering, who had just recognized it in an epizootic state, published a monograph of it, in which, after detailing the results of experiments of inoculation, he studies the differential diagnosis of this disease and acute glanders. About the same period König also described and designated it under the name of *aphthæ ulcerosæ*; he considered it as a contagious exanthema. In 1863 Vimercati mentioned it under the title of *epizootic disease of the mouth* (maulseuche) of the horse. In ninety new army horses, twenty were affected by dermatitis, stomatitis, rhinitis, and pustular conjunctivitis. The publications of Palat and Silvestri upon "variola of the horse" probably relate to the same affection. Silvestri classifies the *impetigo labialis* of German veterinarians with the morbid condition designated by him under the name of "variola." The observations of Sondermann, Immelmann, Eggling, and Haarstick refer also to pustular stomatitis. Finally, Eggling and Ellenberger have made new researches upon the disease in question, and published a work in which they designate it under the name of *contagious pustular stomatitis*.

Etiology. As indicated by its name, this stomatitis is extremely contagious. Its virus is fixed; it exists in the saliva and the buccal

mucus; the mucous membranes are much more sensitive to its action than the skin. Usually it penetrates into the organism through a wound; in the horse, however, infection may be the result of simple contact. If considered in the order of receptivity for the virus of pustular stomatitis, the various species are classified in the following order: horse, ox, man, sheep, and pig (Eggeling and Ellenberger). Friedberger has transmitted the disease to the chicken. Contamination of man occurs usually through the hands or arms.

The virus seems to become attenuated by inoculation made in series upon animals (Eggeling and Ellenberger, Friedberger). Its nature is as yet unknown.

From an etiological standpoint pustular stomatitis has nothing in common with *dermatitis contagiosa pustulosa*.

Pathological anatomy. According to Eggeling and Ellenberger, the initial alterations consist of inflammatory cellular infiltration of the papillary body. Macroscopically they appear in small papules; these soon undergo purulent disintegration in their centre; the papillæ are destroyed. The process may involve the whole thickness of the skin or the affected mucous membrane; it may even extend to the subcutaneous or submucous connective tissue. Later, when the pustules thus formed open, the zone which surrounds them is also the seat of inflammatory infiltration. The ulcerations become filled with granulations; the epithelium is reformed from the periphery to the centre of the cicatrices. Complete recovery requires but twelve days.

These alterations, which are sometimes isolated and sometimes confluent, are especially found in the buccal cavity upon the internal face of the lips, upon the sides of the mouth, the point and lateral face of the tongue. In some subjects they are also seen upon the mucous membrane of the nose and upon the skin of the neighboring regions.

When the process invades the conjunctiva it generally produces a purulent catarrh. In some rare cases the pustules developed upon the mucous membranes are preceded by red specks (Friedberger).

Symptoms. The disease is marked from the onset by slight fever (a temperature of 39.5° C. and 60 pulsations), and by abnormal redness and heat of the buccal mucous membrane. On passing the finger upon the affected surfaces we perceive a few isolated

papules; ptyalism may be soon observed; abundant mucus accumulates in the mouth. The animals resist any exploration of that cavity. In the beginning the general condition is not impaired very much, and the appetite is preserved; later, mastication and deglutition become very painful—they are accompanied by abundant salivation and regurgitation. We rarely observe a high temperature (40° to 41.5°). At the end of two to three days the papules, which have become more numerous and larger, are white upon their apex; they are transformed into pustules and have become ulcerated. From the fifth day the buccal mucous membrane is covered with ulcerations in various stages of development. Upon the inner face of the lips, tongue, and gums we find isolated or agminated (gathered in groups of two to seven and sometimes more) conical prominences 2 to 3 millimetres in height, the crest of which is transformed into a small circular ulceration, which presents a hyperemic, shiny, finely granulated central depression; this ulceration is surrounded by a whitish-gray or yellowish-gray ring having the aspect of parched epithelium, easily detached and covering an ulcerating surface, bleeding at the slightest contact, and similar to that of the central portion. Besides these lesions there are wounds formed by the confluence of a certain number of pustules, and which often extend as far as the subcutaneous connective tissue.

Similar alterations may be seen upon the skin of the upper lip, cheeks, around the nostrils, upon the wall of the nose, and at regions more distant from the head—upon the front legs, for instance.

The cicatrization of the wounds frequently begins as early as the eighth day. The duration of the disease depends upon its intensity. It lasts on an average fourteen days, or three weeks at a maximum.

Besides these local affections we find the pituitary and the conjunctiva red and infiltrated; the ocular mucous membrane is at times the seat of an intense purulent catarrh. The lymphatic ganglions, especially the submaxillary, are tumefied; prescapular obstruction of the ganglions has sometimes been observed.

Differential diagnosis. Although it is quite difficult to make the diagnosis of pustular stomatitis, we may, however, be able to establish it with certainty. In all cases we have especially to differentiate it from the following diseases:

1. *Acute glanders and farcy.* Pustular stomatitis has often been confounded with this latter affection. Thirty years ago Hering

established the basis of the differential diagnosis. Mistakes are particularly easy to make when there exist at the same time ulcerations of the pituitary and of the skin, a nasal discharge, a hard, painless tumefaction of the lymphatic ganglions, and when contagion is manifest. It is to be supposed that at the period when contagious stomatitis was unknown many horses affected by this disease were considered as glandered. The following indications enable us to make a distinction.

a. Pustular stomatitis has a mild course.

b. Its clinical symptomatology is different from that of glanders and farcy.

c. In cases of pustular stomatitis with cutaneous ulcerations, these are not located exclusively along the lymphatic vessels, like chancres of farcy; they are not arranged in moniliform series, and their border is not cut sharp, indurated and phagedenic. Scattered and of circular form, they are generally found covered with a solid brown crust; their fundus is overspread with a layer of granulations; upon their borders we observe an active epithelial neoformation; finally they heal within from eight to twelve days.

d. Ulcerations developed upon the pituitary are located exclusively in the neighborhood of the nostrils; they are distinguished from glanderous chancres by their characters, which are similar to those of cutaneous lesions.

e. Inoculation produces pustular stomatitis, never glanders; in the ox, especially when it is practised upon the vaginal mucous membrane, it almost always succeeds.

2. *Variola of the horse.* This disease offers most analogy to pustular stomatitis; the principal diagnostic symptom is the difference of localization: horsepox is mostly seen in the pastern fold.

3. *Follicular ulceration.* It was formerly connected with contagious pustular stomatitis, but it has nothing in common with this latter disease as far as its nature is concerned. In pustular stomatitis the ulcerations are not located exclusively on the surface of the follicles; they are also seen in points where no glandulæ exist.

4. *Labial herpes.* During the course of this disease we see neither pustules nor ulcerations.

5. *Catarrhal, aphthous, traumatic and ulcerous stomatitis.* They are not propagated upon the nasal mucous membrane, and do not lead to tumefaction of the lymphatic ganglions. In their evolution

they do not possess the typic regularity of pustular stomatitis; finally they are not transmissible.

6. Coital exanthema and dourine hardly deserve to be mentioned, for up to date the characteristic alterations of pustular stomatitis upon the genital mucous membrane have only been observed after experimental inoculation(?).

Prognosis. It is not serious. The disease has never been fatal.

Treatment. Like all acute exanthema, this stomatitis follows a typic course; the *morbid indication* is therefore wanting. The symptomatic treatment consists in the application of lotions upon the ulcerations, consisting of disinfecting solutions: cresol, alun, sulphate of iron, chloride of potassium, etc. The disease is very contagious; it is important to take prophylactic measures.

DIPHTHERIC DISEASES OF DOMESTIC ANIMALS.

GENERALITIES UPON THE TERM DIPHTHERIA. We give the name of diphtheria to a particular form of inflammation of the mucous membranes characterized by a fibrinous exudation which takes place in the substance of the tissues and which leads to their necrosis, or by a mixed process, exudative and necrotic. An essential anatomical fact distinguishes diphtheric and croupous phlegmasias; the latter is superficial, it produces the formation of fibrinous membranes, which easily become detached, but it never invades the deep layers of the mucous membrane.

Diphtheria of the mucous membranes, such as we have defined it, does not constitute a clinical entity. Its causes are multiple. It may be produced under the influence of mechanical pressure (in coprostaia, for instance). A large number of chemical substances, but particularly caustics, may determine diphtheric necrosis of these membranes. Similar processes are generated by numerous infectious agents. In domestic animals they are observed during the course of bovine plague, malignant catarrhal fever of the ox, swine plague, acute glanders, petechial fever, the "sniffing disease" of the pig, puerperal septicæmia, and gregarinosis of poultry. By the inoculation of certain microbes, Heubner experimentally produced in animals local diphtheric inflammations.

In man diphtheria, when considered from a clinical standpoint, is quite as variable in its nature. Physicians have, therefore, for a long time divided it into *primary* and *secondary*. Primary diph-

theria, or simply *diphtheria*, is an infectious disease which is autonomic, and especially characterized by specific inflammation of the mucous membrane of the back of the mouth. But diphtheric anginas are also observed in the course of a large number of other infections, scarlatina, measles, variola, erysipelas, whooping-cough, etc.; these latter bear the name of *secondary diphtherias*.

GENERALITIES UPON ANIMAL AND HUMAN DIPHTHERIA. We have already affirmed, in the first edition of this work, that on many occasions the diphtheric diseases of animals have no relation with the diphtheria of man. No transmission of the latter to animals, with its specific infectious qualities, has ever succeeded; on the other hand, there does not exist any authentic observation of contamination of man by animals affected by diphtheria. If we examine attentively the value of "cases of transmission of chicken diphtheria to man," we recognize that they are based upon probabilities or upon simple hypotheses, which can only be explained by complete ignorance on the questions which pertain to veterinary pathology. Almost all have the stamp of error. Only that of Gerhard, in which two-thirds of persons having charge of chickens affected by diphtheria (in a breeding establishment) were attacked with diphtheric angina, deserves mention; but this fact mentioned by Gerhard was not observed by himself. If contamination of man by chickens was possible, numerous instances would be found, on account of the frequency of diphtheria in these birds. We have examined hundreds of diphtheric chickens, without ever observing any case of contamination among us or the people who had charge of the patients. Domestic animals are not affected by any infectious disease identical to human diphtheria, and, from an etiological and clinical standpoint, diphtheria of the chicken is essentially different from that of man.

Besides, as we have just stated, this latter could never be transmitted experimentally to animals. The inoculations made by Collin in the pig, by Harley in the dog, by Pentzold in the rabbit, chicken, and pigeon, and by Esser in the calf, have all remained without result. Löffler has not succeeded any better in transmitting it to animals, and his bacteriological researches have established that the inflammatory products of the diphtheria of man, and aviary and vitular diphtherias, contain different micro-organisms.

Oertel, Jaffe, Letzrich, Nassiloff, Eberth, Thomasi, Frisch, Trendelenburg, and others who claim to have obtained positive

results, have really only transmitted necrotic diseases of the mucous membranes and acute infections of the blood.

The experimental production of a diphtheric inflammation of the mucous membranes would besides be insufficient to establish the identity of these processes; diphtheric phlegmasia of these membranes may indeed be produced by very different micro-organisms. There has never been produced any clinical tableau similar to human diphtheria by inoculations to animals. Emmerich claimed to have met the same microbe in diphtheric products coming from man and pigeons; but this assertion requires investigation, and so much the more because at the present time the infectious agent of human diphtheria is not yet determined.

Diphtheria of man is an acute infectious disease generally localized in the posterior part of the larynx, where it is marked by a specific inflammation of the mucous membrane. The agent of this infection is still unknown. Löffler considers it to be a bacillus; Oertel, an ovoid microbe; Emmerich, a bacterium; Aufrecht, a micrococcus, which is also found in the lesions of croupous pneumonia and puerperal fever. All these authors consider the microbe which they have discovered as the pathogenic agent of diphtheria. This affects in preference children (up to the age of ten years). It is extremely contagious, and seems to start by a local disease of the throat. After a period of incubation of from two to five days we observe general troubles (fever, general uneasiness, headache) and dysphagic symptoms. The mucous membrane of the velum palati is red, the amygdalæ are tumefied; we soon see the appearance upon the mucous membrane of the throat (tonsils, uvula, and arch of the palate) whitish-gray exudates, which are very adherent, and also great tumefaction of the neighboring lymphatic ganglions. Later the process invades the mucous membrane of the larynx and produces a contraction of this canal (dyspnœa, suffocation); it may be propagated to the mucous membrane of the bronchi and bronchioles. We observe besides the symptoms of general septic infection. As complications we recognize the extension of the diphtheric inflammation to the nasal, buccal, and intestinal mucous membrane, to the cavity of the tympanum, to the conjunctiva; also articular tumefactions, myocarditis, nephritis; paralysis of the velum palati, vocal cords, ocular and pharyngeal muscles, more rarely of the muscles of the legs; finally, ataxia. The mortality is very high.

I. Croupo-diphtheric Inflammation of the Mucous Membranes in Domestic Poultry.

GENERAL CONSIDERATIONS. Diphtheria of birds does not constitute a simple morbid species from an etiological standpoint. We must, in fact, recognize in it two forms, which are nearly similar in their symptoms, but essentially different in their nature:

A. Croupo-diphtheric inflammation of the mucous membranes, very probably of microbial origin.

B. Croupo-diphtheric inflammation of the mucous membranes, produced by gregarinæ.

The first has special anatomo-pathological characters; besides, notwithstanding its subtle contagiousness (cohabitation), it is very difficult to transmit it by inoculation. Very few experiments have given positive results (Trincherà). On the contrary, gregarious diphtheria may very easily be inoculated.

We might establish a third group in aviary diphtheria, comprising inflammations, which are produced by *Cercomonas*. In the deep layers of yellowish exudates coming from chickens affected by croup of the pharynx, of the œsophagus and crop Rivolta has found thousands of ovoid infusoria, which he considered as the essential agents of pseudo-croupous inflammation, and to which he has given the name of *Cercomonas gallinarum*. Zürn has found the cercomonas in the pigeon; they had produced a slight inflammation and a yellowish gelatiniform exudate slightly adherent to the mucous membrane.

We cannot agree with Zürn's opinion concerning the identity of both forms which we have just established in cases of diphtheric inflammations. This author admits that in diphtheria which is produced by gregarinæ these disappear after having given birth to micrococci and bacteria which are capable of producing microbial diphtheric inflammation. We would thus not have to deal with two morbid species, but only with two different and successive periods of evolution of the same disease. In microbial diphtheria we have never found any gregarinæ, notwithstanding that they may be very easily recognized. Quite recently Pfeiffer has established a new theory, according to which diphtheria of the chicken is said to be produced by flagellés (protozoaires). The diversity of the anatomo-pathological characters of the disease is due to the fact that they may be observed at different periods of the develop-

ment of the flagellés. The rôle of the microbes would be entirely accessory. We cannot indorse Pfeiffer's theory. It has been impossible for us to discover the protozoaires which are incriminated by this author in the various phases of microbial diphtheria.

A. CROUPO-DIPHTHERIC INFLAMMATION OF THE MUCOUS MEMBRANES, VERY PROBABLY OF MICROBIC ORIGIN.

Etiology. This first principal form of diphtheric inflammation of the mucous membranes in its most comprehensive acceptation constitutes, after cholera, the most destructive epioritic disease of our bird species. It has only been known since 1850, when it was pointed out by Leisering. According to Zürn, Russ, in 1861, wrote the first description of it. The numerous publications of Italian and French veterinarians, as well as certain etiological facts which will be given later, seem to indicate that Italy and France represent the principal and primary centres of this devastating epioritic, which was imported into Germany.

Some authors thought at first that it was of tuberculous nature; later they considered it as *true croup* or *true diphtheria*, as a croupo-diphtheric inflammation. When various microbes had been found in the inflammatory products they attributed to them a pathogenic rôle. Quite recently also several micro-organisms were accused, without giving the demonstration of their specific activity. For chicken diphtheria Rivolta incriminated two particular species of fungi (*Epitheliomyces croupogenus*), which are developed upon the skin and the mucous membranes; they vegetate neither in the tissues nor in the blood—a fact which may be opposed to coccobacterial infection of the blood in human diphtheria. In diphtheric pigeons, in exudates, and in the liver, Löffler has found, besides multiple microbial species, little rods with rounded extremities which are a little longer and thinner than those of septicemia of rabbits. He has been able to cultivate them. By a subcutaneous injection of the cultures he has produced necrotic inflammations. In the mouse he has determined an infectious process with typical characters, and in two pigeons, with a culture obtained by cultivating hepatic tissue of an infected mouse, he produced a specific inflammation of the buccal mucous membrane. However, Löffler not being able to determine the constancy of these little rods in the

diphtheric lesions of the pigeon, has not formulated any strict conclusion concerning their pathogenic rôle.

AFFECTED ANIMALS. The disease exists almost always in an epiornitic state. It mostly affects chickens and pigeons of improved breeds which are of Italian or French origin. In Germany indigenous chickens are little affected by it. Young birds are more exposed to it than old ones. Besides the gallinaceous breeds (chicken, turkey, peacock, pheasant) and pigeon, it is also observed in parrots and palmipedes.

The great extension of diphtheria in recent times is especially due to the numerous importations of improved foreign breeds. Frequent exhibitions of poultry (agricultural fairs), during which the necessary prophylactic measures are not always observed, contribute also in great measure to its propagation.

Symptoms. The clinical picture of microbic diphtheric inflammation of the mucous membranes is extremely complex and polymorphic. It depends especially upon the seat of the affection. This may take place upon the mucous membrane of the mouth, pharynx, upon the pituitary and the membrane covering the sinus, upon the mucous membrane of the laryngeal pouch and the conjunctiva; from the buccal cavity it may extend to the larynx, to the trachea and bronchi, and to the pulmonary alveoli; in some cases it becomes fixed upon the intestinal mucous membrane; in others it invades the skin. These localizations are found isolated or coexisting.

1. *General symptoms.* In mature and strong animals the general condition is unimpaired or but little altered in the beginning. Very often the local alterations are already well advanced when the condition of the subjects attracts attention (contrary to what is observed in diphtheria in man). Hyperthermia is never in proportion to the extension of the local process; this is an observation which we must emphasize, for the opposite has been affirmed.¹

Later we observe symptoms of circulatory troubles and of decrease of the general nutritive condition, then a wasting away, with its usual complications. The crest and the maxillary lobes (barbillons)

¹ In seven perfectly healthy domestic chickens we have observed a temperature varying from 41.7° to 42.3° C.; in two others it oscillated between 42.4° and 42.6°. Munk, in his "Physiology of Domestic Animals," gives as normal temperature of the chicken 42°. In diphtheric chickens Trinchera has constantly found a temperature between 41.6° and 42.5°.—N. D. A.

are generally dry, sometimes hot and sometimes cold; the mucous membranes are pale and the feathers erect. The patients stop eating and laying; they are depressed, weak, let their wings hang, they keep by themselves, and may be caught without difficulty or attempting to run away. Toward the end the general temperature is decreased. At certain moments we may observe symptoms of cerebral excitement which are soon replaced by deep coma.

2. *Diphtheria of the mouth and throat.* In its beginning this localization generally passes unperceived. We may then observe hyperemia and slight tumefaction of the affected mucous membrane. Soon (sometimes within twenty-four hours) this becomes covered with a coating which is similar to frost; this covering becomes rapidly thicker, more resistant, shiny, caseous; afterward it takes the aspect of a pseudo-membrane which may acquire a thickness of 1 mm. to 1.5 mm.; it is dirty yellow in color, and later brownish; its consistency is dry, friable; later it becomes rugous on its surface and cracked.

Ordinarily the neck and the head are stretched; the patients breathe with the bill open. Prehension and deglutition are more or less difficult.

The false membranes are not uniformly disseminated upon the invaded mucous surfaces. They are mostly located upon the palate and its velum, on the lower fascia, the frænum, and the point of the tongue, upon the edges of the bill, cheeks, and the upper part of the larynx. From the edges of the bill the affection sometimes extends to the skin.

Fresh diphtheric exudates are very adherent to the subjacent mucous membrane; in removing them a more or less deep ulcerating wound is laid bare, which is irregular, red, bloody, having borders which seem knawed; also a granular bottom strewn with fine villi; their deep layer is penetrated by vascular loops; often in pulling these off we tear bloodvessels and provoke a slight hemorrhage.

At a more advanced period these exudates may become detached. Upon their surface the mucous membrane is sometimes intact; sometimes it presents chancre-like ulcerations, deep losses of substance, even necrotic destruction in some regions (point of the tongue, for instance).

3. *Diphtheria of the pituitary and of the membrane which covers the accessory cavities of the nose.* In the beginning a serous liquid

runs out of the nostrils, which afterward becomes viscous, dirty-yellow; in drying up it partially obstructs these orifices and the nasal cavities. If we compress one of the cartilaginous wings of the nose, we produce a trickling of serous, mucous, milky, or purulent liquid from the other nostril, according to the period of the affection. Breathing is laborious and wheezing; the patients shake their head frequently and are seized with an attack of sneezing, expelling masses of mucus. The fissure of the palate is sometimes filled with diphtheric productions.

Very frequently the morbid process becomes propagated to the covering membrane of the infra-ocular sinus. Ordinarily this inflammation of the sinus exists on but one side. Above the internal angle of the eye and behind the root of the bill we observe a swelling of the soft parts, which are hyperemic, œdematous (collateral œdema). This region is hot and very sensitive to pressure; there is a discharge from the corresponding side. In the course of time tumefaction increases; it extends to the ocular globe and backward; finally it may acquire the size of half a walnut. The corresponding palatine bone becomes hypertrophied, especially in a transverse direction; it protrudes markedly on the interior of the buccal cavity. At a more advanced period the infra-ocular region is much distended and hard. When it is explored in time we find a creamy, thick, whitish liquid or yellowish and soft caseous masses. Later its contents are dry, grumous; sometimes it is arranged in thin, concentric layers, or it forms more or less voluminous collections; it may become very abundant and lead to considerable dilatation of the sinus, the diameter of which reaches two, three, and even four centimetres. The soft tissues of the neighborhood are crowded back, the ocular globe is prominent, the palate protrudes, the bones are depressed, and the head deformed. The patients can hardly close the bill, and take but little food.

4. *Diphtheria of the larynx and trachea.* We often find diphtheric productions in the larynx, trachea, and in the surrounding tissues. In poultry, especially in large chickens, it is easy to explore these regions. According to the thickness of the false membranes and the degree of contraction of the larynx, we observe the symptoms of a more or less intense dyspnoea; the respiration is deep, laborious, sometimes panting; the bill is wide open; we hear particular wheezing or quivering bruits, which are accompanied by moist râles and cough. The expectorated exudates accumulate in

the throat; then they are ejected through the opened bill and soil the front part of the body. By becoming decomposed they produce a nausous, disagreeable odor, which is perceptible at some distance from the patients. Death may occur by asphyxia.

5. *Ocular diphtheria*. In the eyes the affection begins with the symptoms of conjunctival phlegmasia. Hyperemia of the initial stage is soon accompanied by muco-purulent catarrh; secretions accumulate in the internal angle of the eye, and then run out. The eyelids and neighboring tissues are cedematous and hot. We often find the velum of the lids agglutinated; in parting them we give exit to serous, inflammatory, purulent, or caseous products; these latter are moulded upon the ocular globe, and show a semi-lunar or lenticular form.

When the trouble is left to itself the eye becomes atrophied under the permanent pressure of the exudate, where it is overrun by the diphtheric process, which rapidly produces serious destruction (panophthalmia). From the sclerotic membrane the phlegmasia at first extends to the cornea, which becomes dull, cedematous (parenchymatous keratitis), and frequently covered by an exudate of conic form, which increases rapidly in thickness and separates the eyelids. The cornea suppurates and becomes perforated. In most cases a purulent panophthalmia is developed, which leads to atrophy of the ocular globe.

6. *Intestinal diphtheria*. In the intestine diphtheria produces catarrhal inflammation, which is more or less intense, and also an extensive infiltration of the mucous membrane. In general this intestinal affection appears late; it is preceded by other localizations; it is the last stage of the process, the terminal link of the symptomatic chain. However, according to Zürn, it occurs very soon upon palmipedes and turkeys. It is marked by aggravation of all the general troubles, by a fetid, serous, mucous, purulent, or hemorrhagic diarrhea. This intestinal flux leads to insensibility, apathy, and exhaustion. When it becomes very abundant death is near (Zürn).

7. *Cutaneous diphtheria*. The extension of microbic diphtheria upon the skin occurs only in the neighborhood of the natural orifices (around the eyelids, the edges of the bill, the anus, and the external auditory canal). In these regions we may observe on the integument losses of substance by ulceration. Tuberculiform neoformations of the skin belong to the domain of gregarinous diphtheria.

Pathological anatomy. The cadavers are anemic and emaciated. Besides the lesions observed during life we may find, accumulated in the bronchi, yellowish-white masses, which are half liquid, or solid granular matter; the bronchial canals are sometimes completely obstructed; the pulmonary parenchyma is actelectasic or œdematous. There often exists a tracheo-bronchial catarrh and diffuse catarrhal pneumonia. Cellulo-fibrinous pericarditis, with sub-pericardiac ecchymoses and parenchymatous tumefaction of certain organs (liver) are not rare alterations. Catarrhal enteritis is usually localized upon the anterior portions of the intestines. Zürn has found the cæcum and the rectum filled with a yellowish exudate, disposed in concentric layers, and much adherent to the mucous membrane; this is often the seat of ulcerating destruction.

All exudates have the same microscopic constitution; that of the lachrymal pouches is caseous, soft, and principally formed by pus corpuscles, round cells, which are similar to leucocytes undergoing degeneration; these elements, the dimensions of which vary from 6 to 9 μ , are rapidly destroyed. Besides, we find in it numerous microbes, and especially *cocci*. The solution of methylene-violet permits us to recognize in considerable quantities small uniform micrococci. The bacteria seem to be much less numerous. Fresh exudates coming from the mouth and throat offer the same histological peculiarities; desquamated pavement epithelium is, however, more abundant. Those of the sinus and bronchi possess also the same composition. In all diphtheric productions, even in the recent false membranes, the round cells are quickly transformed into a fine granular detritus, which explains the variable microscopic characters of the exudates, according to their age.

Course. The course of diphtheria of the mucous membranes is slow, chronic, and death occurs generally only after several weeks, or even a few months.¹ The experiments of inoculation which have given positive results confirm this assertion. By using nasal mucus, Trinchera observed a latent period lasting from seven to

¹ The experiments which were made by Prof. Colin upon aviary diphtheria (1883-84) have taught that the duration of this affection sometimes lasts two years (Receuil vét., 1885). Mégnin has recognized that the œsophageal diphtheria of the pigeon is compatible with all the appearances of health. This fact explains the perpetuation of this disease in pigeon houses. Parents themselves transmit it to their young by passing into their bill the substances which constitute their first alimentation. (Compt. rénd. de la Soc. de Biologie, 1891.)

twenty days, then the symptoms increased slowly in intensity, in order to reach their maximum in eight to fifteen days. Strong animals recovered within about two months. Young chickens and pigeons are an exception to the rule; they may die very rapidly.

Prognosis. In general, the prognosis is serious. However, for the pigeon and domestic chicken, which are strong and well fed, it is relatively favorable; we may say the same thing when the affection remains localized in the posterior part of the mouth. As a rule, cure is hard to obtain; frequently it is only apparent; morbid centres subsist in which the trouble continues its evolution. This fact permits us to understand how birds which seem cured and are put back among others contaminate the latter. The epiornithic may also become propagated insidiously. The mortality is 50 to 70 per cent.

Treatment. The prophylaxis plays the most important part. It advises the examination of all newly-bought animals and to keep them isolated for several days, and not to take part in any exhibition of birds which are not subjected to sanitary control. We must, besides, observe attentively the various subjects of infected aviaries, in order to find out the first symptoms of the trouble (nasal discharge, watery eyes, etc.), so as to be able to recognize diphtheria in its beginning. We must separate healthy birds from the sick, and keep the premises perfectly clean; the birds should be treated from time to time with phenic acid, sublimate, cresol, lye, etc.; the cadavers should be destroyed and the utensils used in chicken yards disinfected.

When the disease has been recognized it is important to combat it without delay. Constant care and perseverance constitute the most important conditions of success. The number of medicinal agents recommended is considerable. Among them disinfectants come first: Phenic acid, creosote, tar, cresol, chloride of potassium, chloride of lime, tincture of iodine, salicylic acid, boric acid, sulphate of iron, a solution of perchloride of iron, tannin, essence of turpentine, caustic potash, nitrate of silver, lactic acid, etc. All these agents may be used separately or combined.

Sublimate at 1-2 per 1000 and cresol at 1-2 per cent. have appeared very efficient in cases where the process is localized. The loosening of false membranes is only advisable when they are slightly adherent, if the operation may be made without producing hemorrhage (pigeon). We cannot recommend extraction of

exudates and the use of caustics; these methods are almost always harmful. The tumors of the sinus are incised and the hemorrhages stopped with a tampon of cotton dipped in a solution of perchloride of iron. Fumigations of tar have given good results in the treatment of affections of the respiratory passages. Zürn advises, *internally* and *externally*, the following mixture: a decoction of leaves of hazelnut (15 grammes per litre of water), 150 grammes; glycerin, 20 grammes; hydrochlorate of potassium, 5 grammes; salicylic acid, 0.5 gramme dissolved in 15 grammes of rectified alcohol. For large birds we give, daily, 1–2 teaspoonfuls or tablespoonfuls, according to their size; to the pigeon $\frac{1}{4}$ to $\frac{1}{2}$ of a teaspoonful; besides, two or three times a day we make local applications upon the diseased regions with the same solution. To combat intestinal affection we advise tannin or sulphate of iron (in a solution of 1 to 2 per cent. or in pills which are made of bread and butter); the doses are: for the pigeon, 0.3 gramme to 0.6 gramme; for the chicken, 0.6 gramme to 1.2 grammes; for the goose, 0.6 gramme to 2 grammes.

Richards' remedy is composed as follows:

Chloride of potassium	7.5 parts.
Salicylic acid	1.5 “
Glycerin	15.0 “
Plain green syrup	130.0 “

To be applied upon the false membranes of the mouth with a brush.

B. DIPHTHERIC INFLAMMATION OF THE MUCOUS MEMBRANES PRODUCED BY GREGARINÆ (COCCIDIES).

Etiology. Gregarinæ or Psorospermies (also called coccidies when they are encysted) are very small protistes formed by a sarcodic corpuscle. They are of extremely variable form (rounded, spheroidal, ovoid, elliptical, etc.), have amœboid movements in their early stage; later, they become encysted. They are very common parasites of animals, and are found in chickens, rabbits, rats, dogs, fish, snails, and lumbrici, etc. They always exist in innumerable quantities (whence the name Grégariæ; see Vol. I., page 306). Their development has been studied by Eimer and Leuckart. In the first stage of their evolution they are located in the epithelial cells. In poultry they penetrate into the epithelium of the mucous membranes, where they produce serious nutritive troubles which may lead to necrosis of the affected parts. At the microscopical

examination they offer a great resemblance to the large cellular nuclei.

The diphtheric gregarinous processes are distinguished from microbic diphtheric inflammation by their frequent extension to the membrane of the head, by almost constant positive results which are given by inoculation, and by their relatively easy curability when they evolve upon the mucous membranes of the mouth and throat, of the upper portion of the larynx, and upon the skin.

Rivolta and Silvestri were the first, in 1872, who found the psorospermies (naked gregarinæ) in the chicken, during the course of an epiornitis which existed in the neighborhood of Pisa.

Symptoms. Upon the mucous membranes of the head the clinical characters of gregarinous diphtheria are similar to those of microbic diphtheria. The disease consists essentially in a specific phlegmasia of the mucous membranes of the mouth and throat, of the nasal cavities, the larynx, and the eye, etc., accompanied by secondary intestinal affection. We may also observe primary gregarinous enteritis (Zürn).

But gregarinous diphtheria is differentiated from the microbic form by the frequency of alterations of the skin, especially by the appearance on this membrane of tuberculiform vegetations to which we give the name of gregarinous epitheliomata (*epithelioma gregarinosum* of Böllinger, which is identical with the *molloscum contagiosum* of man). These epitheliomata preferably develop on the head and on the regions deprived of feathers (roots of the bill, edges of the buccal opening, auricular lobules, around the external auditory canal, maxillary lobules, the face, eyelids, comb, and throat). Sometimes they extend to the feathered portions of the head, neck, and to the cervical stem; they may also be found upon the external face of the legs, abdomen, under the wings, and in the neighborhood of the cloaca.

These tumors have at first the form of flattened tubercles; later they become prominent; in the beginning their volume is nearly that of a poppy-seed or a millet-seed. Their shade varies from clear red to grayish-yellow. When very young they possess a fatty, pearly reflex; in general they are quite hard to the touch, but their surface soon becomes covered by a dirty-gray, brownish-yellow, or brownish-red crust. They are found in variable numbers and developed in various regions, particularly upon the erectile organs. Their dimensions vary with their age. Sometimes the affected sur-

faces are granular ; at other times they offer a warty appearance, and are covered with proliferations which have the shape of myrtle-berries. According to their isolated or confluent condition, these vegetations may have the size of a lentil, a pea, cherry-stone, or a bean ; we find some that are much larger in size. The diseased parts become more rugous and irregular as the lesions increase in age.

When these pseudo-tubercles are developed upon the eyelids they become infiltrated, thickened, and agglutinated. The conjunctiva is generally affected ; it is tumefied, projects between the palpebral wings, and presents the symptoms of catarrhal inflammation ; at the points where the eruption takes place there is a yellowish coloration covered with crusts. Later the phlegmasia becomes purulent ; if it extends to the cornea and to the sclerotic membrane, it produces a keratitis and a panophthalmia ; when it is propagated to the external integument of the eyelid and to the adjacent regions, as sometimes happens in the pigeon, the whole eye is covered by a neoformation, more or less voluminous, presenting the aspect of a myrtleberry.

Course and prognosis. In general, gregarinous diphtheria is less serious than that which is of microbic origin. Healing often occurs spontaneously ; the vegetations dry up and drop out. The buccal, pharyngeal, and laryngeal localizations frequently heal without any treatment.

However, even in these localized forms death may occur by asphyxia (obstruction of the larynx or of the trachea). In some cases it is determined by cachexia, by eruptions and catarrhal phlegmasia, or by an intestinal affection. We then observe inappetence, depression, and erected feathers. Bollinger has seen chickens die four to five weeks after infection, or three to four weeks after the beginning of the eruption.

Pathological anatomy. When we examine diphtheric productions or their detritus we find therein desquamated epithelial cells containing a round corpuscle, which fills more than half of the cellular cavity (gregarina). This homogeneous and much refringent corpuscle, with fatty or mucous reflex, has been considered as a hypertrophied nucleus. In all diphtheric exudates we find similar spheroidal productions.

An examination of the diseased mucous membrane reveals an identical cellular alteration, when the opaque exudate and necrosis

of coagulation of the anatomical elements permit us to recognize it. All the epithelial cells contain the small rounded body of which we have just spoken. The same is the case with hypertrophied and crowded elements which constitute the substance of epitheliomata (alterations which were formerly designated under the erroneous name of "chicken variola").

On the sections made perpendicularly in the substance of an epitheliomatous tubercle hardened with alcohol we observe a considerable quantity of neoformed and very voluminous epithelial cells. All, with the exception of the youngest, contain a rounded or elongated corpuscle, which at first seems to be a hypertrophied nucleus. This element offers a fatty reflex and infiltrated aspect so much the more manifest in proportion to the age of the lesion. In the very young cells it has the dimensions of a small nucleus its volume is soon doubled or trebled, and toward the superficial epithelial layers it occupies the whole of the intra-cellular cavity; then its aspect is hyaloid to such an extent that the cell might be taken for a large vacuole.

In certain places we observe also elements which undergo proliferation or numerous multinuclear cells. Coloring agents permit us to recognize all these anatomical details. Picro-carmin is more favorable; it colors the cellular nuclei an intense red-brown, while the gregarinæ take a yellow tint.

When we examine a section made perpendicularly on the surface of the mucous membrane and colored by picro-carmin, by studying it from the deep epidermic layer we observe the following peculiarities: the youngest fusiform cells contain an ovoid nucleus, elongated, with one to two nucleoli; in those of the neighboring layer we observe, besides the well-preserved nucleus colored brown-red, a rounded plasmatic corpuscle, homogeneous and colored yellow, which hardly presents the size of a leucocyte, but the dimensions of which increase as we examine cells closer to the other edge of the preparation. The elements thus invaded are distinguished from those which are yet healthy by their considerable size and their distention. Generally the submucous connective tissue is hyperemic and infiltrated with embryonic cells.

Treatment. It is the same as for bacillary diphtheria. We have obtained good results by applying to the diseased regions, by means of a brush, the following preparation: Cresol, 5 grammes; glycerin and distilled water, ãã 100 grammes.

Glycerin when used by itself kills the gregarinæ by depriving them of the water which is necessary to their existence. In gregarinous enteritis it may be administered internally (one teaspoonful or tablespoonful dose every day). This treatment gives specially good results in the goose (Zürn).

II. Diphtheria of the Calf.

Etiology. Dammann and several other authors have described, under the name of "diphtheria of the calf," a diphtheric inflammation of the mucous membrane of the mouth and throat observed in the calf and lamb. The causes of this disease are unknown. It is not demonstrated that it constitutes a pathological entity; it presents, on the contrary, a great similarity with the diphtheric form of stomatitis and pharyngitis, as well as with aphthous fever, and it is probable that they have gathered under the title of *diphtheria of the calf* clinical facts which are related to various affections.

Dammann admits the identity of vitular and human diphtheria; he maintains that these two diseases are produced by the same micrococcus; according to him, the organism of the calf and the stables represent centres of human diphtheria, which until then were unknown. This opinion is not based upon any strict scientific fact. As cause of diphtheria of the calf, Vollers incriminates "a stable miasma." Carbonate of ammonia, which is formed at the expense of urea when stagnant urine becomes decomposed, irritates the mucous membrane and favors the infection.

Löffler, who has made bacteriological and experimental researches upon vitular diphtheria, incriminates a bacillus. In the deep layers of diphtheric lesions he has found long undulating bacilli, which are clearly distinguished by their form and their biological properties from bacilli of human diphtheria. He has inoculated the disease to mice.

According to Dammann, the disease is contagious, and inoculable to the lamb and rabbit and transmissible to man. It affects the calf during the first weeks of life. This author explains this fact by the slight resistance of the buccal epithelium in young animals. The period of incubation would be very short, but the contagious germ would preserve its activity for a long time.

Pathological anatomy. The principal alterations exist in the mouth and throat. Upon the tongue, palate, and cheeks we observe

false yellow croupous membranes, which sometimes reach several centimetres in thickness, and are closely adherent to the tissues upon which they rest. In certain regions the process has completely destroyed the mucous membranes and invaded the subjacent tissues (palate bones, muscles of the tongue). The false membranes are formed histologically by accumulated micrococci, by detritus, fibrinous filaments, and leucocytes; we also find the long bacilli of Löffler.

Similar alterations, but less marked, are developed upon the nasal, laryngeal, tracheal, and intestinal mucous membrane. In one case we have found a false diphtheric membrane, which was very thick, in the interdigital space of both anterior members.

We may also find centres of lobular pneumonia with abscess and purulent pleurisy. The spleen is not hypertrophied.

Symptoms. They greatly resemble those of aphthous fever. The animals stop nursing, salivation is abundant, a purulent discharge escapes from both nostrils, and the cheeks are tumefied. In examining the mouth we observe the alterations which we have just mentioned. Later, when complications follow, they are marked by cough or diarrhœa. During the course of the disease the animals become very weak, they remain almost continually down, and rapidly become emaciated. According to Dammann, there is an average degree of fever; but nothing very exact is known upon this question.

Differential diagnosis. It is difficult to clearly distinguish diphtheria of the calf from aphthous fever. According to Dammann, the former affects but very young animals; it is said to be very malignant, and its course more rapid than that of aphthous fever. In a score of patients which he observed almost all died. This fatal ending occurred ordinarily within four or five days. When death was the consequence of pulmonary or intestinal complications it took place within two or three weeks.

Treatment. From a prophylactic standpoint, the patients must be separated from the healthy subjects and the stable carefully disinfected. We must act upon exudates with disinfectants (phenic or salicylic acid, chloride of potassium, etc.). They must be removed when possible and the mouth washed. Dammann advises frequent gargling with a phenicated solution, at 0.5 per cent., or the use of a paste made of salicylic acid and water, which must be applied with a brush; he also advises to give this acid internally.

MUGUET: THRUSH: APHTHA: PARASITIC STOMATITIS.

Etiology. Thrush is a mycosis of the buccal, laryngeal, and pharyngeal mucous membrane, transmissible from man to animals. It is produced by a fungus which was discovered in 1840 by Berg and Gruby, and was formerly designated under the name of *Oidium albicans*. Grawitz has likened this microphyte to the *mycoderma vini*. Recent researches of Plaut have shown that the fungus of thrush is identical with the *monilia candida*, a parasite of the group of fungi. It vegetates actively upon fresh manure of the cow, decayed wood, in milk and saccharine substances. Its mycelium, which is filamentous, contains brilliant round, ovoid or cylindric cells (conidies). Plaut has cultivated it, and has communicated the disease to chickens by inoculation of cultures. Thrush affects children, calves, foals, and poultry.

Pathology. Young age, a weak constitution, gastric troubles, uncleanness, milky and amylaceous alimentation seem favorable to the development of the fungus of thrush. The various causes which hinder mastication and deglutition produce a prolonged retention of the food, and the formation of acids in the mouth favors infection of the mucous membrane which covers this cavity. In the human species we incriminate vomiting; in the child, when this phenomenon occurs, ejected milk remains in the mouth for some time. Martin has observed a chicken which was infected by a sick child.

Pathological anatomy. In chickens the alterations are limited to the œsophagus and gizzard. Upon the mucous membrane we observe at first small white spots which extend rapidly, they become confluent and soon form a white, gray, or yellow covering, which is divided in islands by furrows, and present a croupous, granular, or viscous consistency. When we raise the membranous exudates we find the mucous membrane red or ulcerated. Histologically, they are composed of epithelial pavement, desquamated cells, between which the fungus forms a thick packing of very thin filaments which end in greenish spores, round or elongated; we also find numerous free spores.

Symptoms. Besides these anatomical alterations, thrush of gallinacæ produces phenomena offering nothing characteristic.

The head is swollen, the mouth exhales an acid odor. The patients are depressed; notwithstanding the preserved appetite they lose flesh and succumb. Death is preceded by violent convulsions.

Differential diagnosis. Stomatitis, which is produced by *Oidium albicans*, is hard to differentiate from other inflammations of the buccal mucous membrane, especially of the microbial diphtheric inflammation (in the chicken), and aphthous, pustular, or ulcerous stomatitis (in suckling animals). Errors must be relatively frequent. However, the diagnosis may be established with certainty by microscopical examination.

Treatment. The organism must be strengthened with good food, and, if necessary, we must combat gastric troubles. The buccal cavity should be kept very clean by frequent washing. It is advisable to remove the exudates and to touch the affected regions with Van Swieten's liquor (Plaut).

Addenda to Infectious Diseases.

1. Under the name of *Texas fever*, *splenic fever*, we designate a disease of the ox which exists in an epizootic state in North America. This disease seems to be an epizootic hemoglobinuria; it is certainly not of anthrax character. Having originated from the borders of the Gulf of Mexico, it has spread to the largest part of the States of North America.

Symptoms. There are inappetence and arrest of rumination, hyperthermia, acceleration of the respiration, ptyalism, bloody diarrhea, hemoglobinuria, emaciation, very marked weakness, and a rapid course. Death occurs frequently within two or three days. Mortality 40-90 per cent.

AUTOPSY.* There are hemorrhages in all the organs under the serous and the digestive mucous membrane; small ulcerations of the abomasum, inflammation of the intestinal mucous membrane, hyperemia of the various viscera, a dark-red coloration of the muscles, and a reddish-brown tint of the blood.

2. *Siberian bubonic disease*, or *jaswa*, is a disease of the horse, which is common in Siberia, where it was known during the last century. Most authors, with Blumberg, of Kasan,¹ considered it as a variety of anthrax. Haupt has given a good description of it. It exists particularly in the plains in the neighborhood of the Volga

¹ Blumberg (communication inédite).

and its tributary streams, especially during the summer. It is not transmitted by direct contagion.

Symptoms. Edematous tumefactions appearing ordinarily in the laryngeal region; also intense fever and serious general troubles. Its duration is about one week. The mortality is enormous. Almost all the patients die.

AUTOPSY. Gelatiniform infiltrations, tumefaction of the spleen, gastro-intestinal hemorrhages. This affection offers a certain analogy to the epizootic disease of wild animals, but its intimate nature has not been determined.

3. *African plague of the horse.* In 1876 this disease existed upon horses, mules, and donkeys of Syria and Egypt, where it was studied by Villoresi and Apostolides. It is extremely serious.

Symptoms. Intense fever, cerebral depression, yellowish-red coloration and tumefaction of the conjunctiva, petechiæ of the buccal mucous membrane, extreme weakness. Death occurs usually within few hours. Maximal duration: two to three days.

AUTOPSY. Septicemia with generalized hemorrhages and parenchymatous degeneration of the principal viscera.

4. *Intermittent fever (malaria)* has rarely been observed in our domestic animals. In man it represents a miasmatic infectious disease which exists almost everywhere (with the exception of the Polar regions), but more specially in low and swampy countries (Campagna of Rome, Pontius swamps, Sicily, Hungary, countries of the lower Danube, tropics). According to Klebs and Tomasi-Crudeli, it is produced by a sporogenous bacillus. It is marked by febrile attacks which are of short duration and have a regular type (this fever may appear daily, or every second, third, fourth, or fifth day, etc. *Febris quotidiana, tertina, quartana, quintana*, etc.). Besides, we generally observe the symptoms of considerable tumefaction of the spleen. There exist also in this affection quite a series of irregular forms (intermittent, remittent, continuous, pernicious fever; chronic cachexia; intermittent larval fever, which is accompanied by neuralgias). Quinine is the specific remedy.

If we compare these facts with the observations of intermittent fever which have been mentioned in animals, we are inclined to admit the existence of this latter in our domestic species. Besides, inoculability of malaria of man to the rabbit and the dog, the observations which have been gathered in Italy (principal centre of this disease), and the good results which have been given by qui-

nine in numerous cases, testify in favor of this opinion. But most facts which in veterinary medicine constitute the *Bibliography* of this question, have nothing in common with true malaria.¹

5. *Scarlatina* is mentioned in old pathological veterinary works. Authors have evidently mistaken petechial fever for this affection. More recently they have affirmed that scarlatina may be transmitted by cow's milk. Klein states that he has found the same micro-organism (*Micrococcus scarlatinus*) in the lesions of human scarlatina, in the milk and ulcerations of cows which are affected by "scarlatina;" by inoculating calves with cultures of this micrococcus he says that he has communicated scarlatina to them. With condensed milk which contained this contagious germ he transmitted the disease to the calf and the mouse. Nevertheless, it is proper to wait for new investigations upon the bovine origin of scarlatina. We do not possess at the present time any very positive information concerning the existence of this disease in the cow.

6. *Recurrent fever* or *recurrent typhus* of man may be inoculated to the monkey. In this affection the blood contains specific spirilles. In India, Steel² has observed in the horse an epizootic disease during the course of which the blood contains mobile spirilles. This disease could be inoculated to the monkey and dog.

7. *Cholera*, "spontaneous," has never been recognized in animals. The "positive results" obtained by inoculation were no doubt but septicemic or putrid processes. However, Koch has succeeded in infecting the rabbit with comma bacillus after having first induced intestinal troubles, and neutralized, by means of soda, the hydrochloric acid of the stomach.

8. In America and Sicily *yellow fever* has been observed, so it is stated, in the horse and the dog.³

9. *Measles* does not exist in domestic animals. The few cases of "measles of the pig," which are mentioned in our publications, must be related to the affection called *Cysticercus*.

¹ Burke has just published upon *Surra* a work giving the following conclusions:

Surra is a malarial fever which takes two forms, one is serious and the other mild. In India the former is generally observed. The influence of an annual high thermic average upon the frequency of pernicious malaria is well known. No matter what the form of the affection is, the medical treatment is of little efficiency. Preventive measures, especially inoculation, ought to be used. As has been recognized by Evans, *surra* is transmissible by the digestive tract and by inoculation. (R. Burke: General Pathol. of *Surra*, 1891.)—N. D. T.

² Steel, *The Vét. Journal*, 1886.

³ Beauville, *An. in Repertor.*, 1880; Chicoli, *Recueil Vét.*, 1884.

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